PATHOLOGY AND THERAPEUTICS

Oral Pathology and Therapeutics

A systematic presentation of the subject from the standpoint of modern therapeutics.

WITH 116 ILLUSTRATIONS

By

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TO

CHARLES P. PRUYN, M.D., D.D.S.,

who for over fifteen years has been my friend and councillor
in all the affairs of life

THIS BOOK IS RESPECTFULLY DEDICATED BY

THE AUTHOR.

Preface.

In nearly all branches of dental science progress has been rapid, and especially in the departments of surgery, operative procedures and prosthodontia, but in the field of therapeutics empirical methods of treatment still abide.

This volume is presented with the hope that it may furnish at least a rational scientific basis for the management of many oral diseases the treatment of which constitutes a large part of our professional service. The recognition and prevention of disease and its consequent misery should be the highest aim of all who follow the healing art as a profession.

It has been the aim of the author not to burden the volume with needless pathological or histological detail, but rather to present such phases of these subjects as will furnish a scientific basis for practical therapeutics. Treatment of disease without knowledge of its pathology has and always will be a decided failure.

Although the preparation of this work has been an arduous task coming into a busy professional life, still it has been a pleasure, for the hope of being helpful to the toilers of the profession, as well as to those who are preparing for their life's work, has been the dominating spirit in which the work has been done.

The author wishes to disclaim originality for much of the subject matter here presented; for the most part it has been gained from recognized authorities, as well as from clinical observations in the college infirmary and private practice extending over a period of nearly twenty years, supplemented by such scientific investigation as a teacher in one of our large schools and a full private practice would permit.

The author wishes to render grateful acknowledgment to Dr. E. S. Willard for personal encouragement and assistance in the work, and to Dr. G. V. Black, Dr. F. B. Noyes, Dr. Martin H. Fisher, Dr. G. W. Cook, Dr. W. D. Miller, Prof. Goadby, Prof. Hopewell Smith, Dr. Osler, Dr. Jas. Nevins Hyde, Dr. E. S. Talbot, Dr. John S. Marshall, Dr. J. Leon Williams, Dr. Hugh Blake Baldwin, Dr. E. C. Kirk, Dr. Sudduth and Dr. Burchard, whose published writings have been drawn on for much subject matter as well as illustrations.

Oral Pathology and Therapeutics.

CHAPTER I.

Dental Caries.

Introductory. History. Recent Theories. Etiology. Bacteriology of Dental Caries. Therapeutics. Curative Methods.

Introductory.

There is no subject in the entire range of dental science that is so important to the dentist as that of caries. The treatment of its ravages constitutes the greater part of his professional labors. The subject will be presented in this chapter from a therapeutic rather than an operative standpoint, although the cure of its ravages most frequently requires operative procedures.

Tooth decay has been prevalent in all ages and among all races of people, but it is only in recent years that its nature has been understood.

Caries of the teeth is a pathological process differing from that found in the tissues in that it is not associated with, or a result of preceding inflammation; dental caries consists in a chemical dissolution of the tooth substance.

History.

In the literature, the names of Boudett and Jourdain seem to be associated with the first scientific movement about 1776, which afterwards led to the theory of decay as a result of inflammation. A little later John Hunter disputed this theory, but advanced no new one.

About the year 1806 Fox offered a further explanation; he held decay was due to inflammation of the lining membrane of the pulp chamber. In 1829 Bell and Fitch held that decay was due to inflammation of the dentine immediately underneath the enamel. Koecker held that not only was the process inflammation of the dentine, but that a second element entered and dissolved the dead portion by means of chemical decomposition.

In 1835 Robertson held that decay was due to chemical decomposition and that inflammation had nothing to do with the process.

Tomes held to this idea and added that there was some disturbance of the dentine, and that the natural resistance of dentine to decay differed according to its vitality.

Here the matter rested for many years, Dr. Watt adding that this chemical decomposition was due to mineral acids developed in the mucous of the mouth. A little later Magitot held that decay was due to chemical alteration in the enamel and dentine brought about through acids developed in the saliva or through agents introduced into the mouth, and

later that putrid decomposition of animal and vegetable substances is responsible for acids found in the saliva.

A little later, about 1867, he brought forward the theory of micro-organic fermentation as a cause for the appearance of acids in the saliva, and that these acids so formed were the direct cause of tooth decay.

Recent Theories.

Here the matter rested until Miller began his remarkable series of experiments, the results of which were published in 1884 and 1885. Professor Miller clearly demonstrated that caries of the teeth is brought about by an acid, probably lactic acid, produced by the growth of micro-organism in the mouth. While Dr. Miller was experimenting and studying in Germany Dr. Black was at work along the same lines in America, and brought out the additional fact that decay is not only the results of acids produced by micro-organic fermentation, but in order to cause decay those acids must be produced at the exact point where decay is to begin.

The profession rather reluctantly accepted this theory, and much controversy has taken place in relation to the manner in which these organisms work. Many have thought that some teeth were more prone to caries than others because of the inherent nature of the tooth; that some were harder than others, richer in lime salts, and that some teeth after calcification undergo a degenerative change, especially in particular spots, which render them more liable to caries.

All of these notions were dashed to pieces when, as a result of experiments accurately made, Dr. Black in 1893 announced to the profession that teeth do not materially differ in this regard, and that our term hard and soft teeth is a misnomer as far as liability to caries is concerned, and that "imperfections of teeth, such as pits, fissures, rough or uneven surfaces, bad forms of interproximate contact points, are causes of caries only in the sense of giving opportunity for the action of the causes that induce caries."—*Black.*

The carious process is slightly different in dentine and cementum from that of enamel. On enamel the organism has to do its work under the influences of a changing saliva, and in the presence of different kinds of food, and is subject to dislodgment by the excursion of foods in masticating, while in the dentine the organisms have a cavity which protects them from most of these influences.

Enamel is harder, has less animal matter, and therefore is more resistant in a certain sense than dentine. The way in which decay of enamel occurs is by the attachment of micro-organisms to its surface, and by their action a dissolution of the cementing substance which holds the enamel rods in place is the first step.

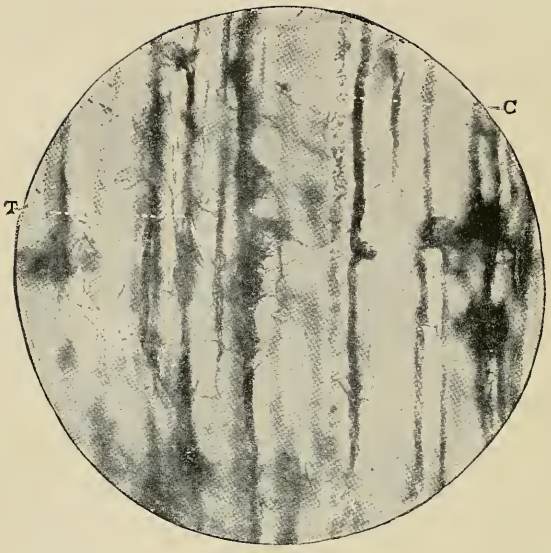

Fig. 1.
Bacilli and micrococci in dental tubules. C, micrococci; T, bacilli. (Hopewell Smith.)

With dentine a somewhat different process occurs. As soon as enamel is destroyed the lime salts around the tubules is dissolved and soon the organisms penetrate the dentine tubules, Fig. 1, and then spread laterally, hence it is that decay passes into the tooth in a somewhat conical shape.

Beyond the field occupied by organisms the lime salts are in a state of decomposition for a considerable distance, which is preparing the way for further ingress of the organisms. As the lime salts are dissolved there is left the animal matrix which furnished food for these organisms.

The profession has been slow to take up these theories of Miller and Black; many objections have been offered, chief of which centered in the thought expressed in the question, how is it possible for micro-organisms to remain in contact with *unbroken enamel* long enough to accomplish its dissolution? This question was finally answered by Dr. J. Leon Williams, of London, England, in 1897. He demonstrated that the micro-organisms that cause tooth decay are gelatine producing organisms. These organisms collect in protected spots about the teeth and form a gelatinous film which is very adherent to the enamel, and under this protection they do their work. Dr. Williams succeeded in grinding specimens thin enough without disturbing the gelatinous plaque over the decaying enamel to show the carious process, Fig. 2. These gelatinous plaques seem to be no bar to the passage of food material for the organism.

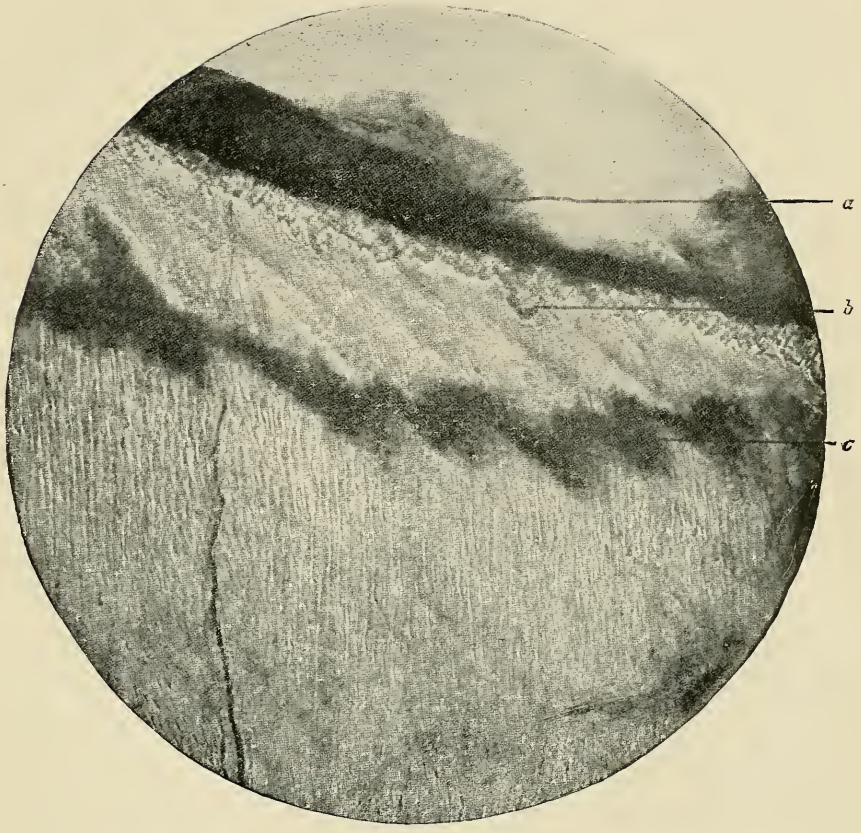

Fig. 2.

From a section of human enamel in early stages of decay. *A*, micro-organisms deeply stained; *b*, evidences of recent vigorous action of acids; *c*, temporary arrest of the organisms. (Williams.)

Etiology.

From a Therapeutic Standpoint.

The present understanding of the causes that have to do with tooth decay are divided into two classes, predisposing and exciting.

By predisposing causes is meant that condition of the general system whereby the secretions of the mouth favor a certain kind of micro-organic growth and development. Just what these conditions of saliva are has not been made out. It was formerly believed that acid conditions favor the carious process, but this has been proven erroneous. Certain it is that the presence of carbohydrates in the saliva favor the process, for they act as food material for the organisms.

The second predisposing cause lies in tooth imperfections which make favorable places for these organisms to take hold and develop.

The exciting cause is acid produced at the immediate point of decay by the action of micro-organisms.

Bacteriology of Dental Caries.

Tooth decay is essentially a bacteriological problem. Without bacteria there would be no tooth decay. They are found everywhere, and especially in the human mouth; here certain forms are constantly found.

It seems that both coccus and bacillus forms have to do with caries, although Miller was formerly of the opinion that only the bacillus form were directly concerned. He isolated four varieties of bacillus, and Goadly classifies three forms of cocci in addition. Black is of the opinion that both varieties have to do with caries, and certainly they are both frequently found in the dentinal tubules, as Fig. 1 illustrates.

Therapeutics.

What has been said up to this point is a brief résumé of the carious process given as a basis for the practical therapeutics of the subject.

The practical therapeutics of this subject can most easily be presented under two heads.

First, Prophylaxis, which relates to the means of preventing or limiting tooth decay. Second, Curative methods, which relate to the arresting of its ravages in a given tooth or several teeth when the carious process has once begun.

The subject of prophylaxis is one of great interest, and although caries has been known for many centuries, still very little has been accomplished in the way of prevention. It is in this direction that scientific work is needed.

Since bacteria everywhere abound, it is not possible for us to prevent them gaining access to the oral cavity, but we are able to hold in check their ravages by three methods.

First and most important relates to the limiting of their food material, which can only be done by limiting the amount of carbohydrates allowed to remain in the oral cavity. Second relates to the cultivation of habits of cleanliness about the oral cavity. Third relates to the use of antiseptics in the oral cavity that will in a measure at least control bacterial activity.

It should be stated that while carbohydrates are essential to proper nutrition, the tendency to consume far in excess of the needs of the system is very great, especially among children. After all, the important point of the matter is not to allow these things to remain in the mouth long enough to undergo fermentation.

The second method.

Many a person is unclean about his mouth because he does not know how to properly care for it and others fall into careless habits. If individuals could be taught to habitually cleanse every surface of every tooth twice daily there would be very little decay. As stated before, these gelatinous plaques cling in out-of-the-way places, and unless an effort is made to cleanse these spots decay will result.

The eating of coarse foods has a tendency to cleanse the teeth by its excursion over their surfaces in mastication, and from this standpoint is valuable. Some think too much brushing injures the teeth, but I have never seen such a case. For prevention of decay, on retiring is the important time to brush.

This subject will be treated further in the chapter on Cleaning Teeth.

The third method, regarding the use of antiseptic mouthwashes, it should be stated that no known remedy can be used strong enough to insure thorough antisepsis. Such a thing as asepsis of the oral cavity cannot be hoped for. There are remedies that would render conditions decidedly antiseptic, but they are so irritating to soft tissue that they can only be employed in attenuated solutions.

Most mouthwashes in the market are antiseptic in their tendency, although the most of them are more adapted to furnishing a pleasant taste than to rendering conditions antiseptic. Conditions of the mouths differ, requiring a little different wash for each case.

The agents that will furnish a basis from which to combine a mouthwash adapted to individual mouth conditions are benzoic acid, borax, carbolic acid, boric acid, oil cassia, trikresol, Black-1-2-3, bichlorid of mercury, hydrogen dioxid, permanganate, wintergreen and other essential oils as flavors.

In the use of an antiseptic mouthwash the patient should not only rinse the mouth but should hold a mouth full of the solution for several minutes.

Curative Methods.

The curative methods are three. First, The removal of all disintegrated tooth substance and polishing the surface. Second, The removal of decay and filling the cavity. Third, The use of medicinal agents that arrest the progress of decay.

The first method is only adapted to those cases where only a slight amount of enamel is disintegrated, and which can be removed without exposing the dentine or impairing the service form of the tooth or its approximate contact. In these cases not only should the softened enamel be removed, but that surface should be made *smooth* and *highly polished.*

Second, The curative effects of fillings depend on three things, that the area of liability of that cavity has been included in the prepared cavity, and that a properly shaped tight filling is made and that no dentine has been left exposed.

The third method relates to the use of such agents as nitrate of silver in shallow, slow forming cavities in deciduous teeth, which will often arrest decay until time for them to be shed. This method is also recommended in those cases where slight decay occurs along the gingival line of molars and bicuspids. Not only will it relieve the sensitiveness which is usually found at those points but will often arrest further decay. Other agents have been suggested, for example, formalin, chloride of zinc, trichloracetic acid.

Recently McKesson & Robbins have suggested a tooth powder of calcium carbonate which will generate hydrogen dioxide when in contact with the lactic acid of decay, which they claim will effectually arrest decay.

CHAPTER II.

The Dental Pulp.

The Functions of the Pulp. Sensitive Dentine. Other Cells. Blood Vessels. Nerve Supply.

Before entering into a study of pulp diseases it seems wise that we hastily reveiw the histology of the tissues involved.

Dental pulp is the name given to the soft tissue occupying the central cavity of the dentine. It is made up of embryonal connective tissue, and many blood vessels and nerves, but no lymphatics. There are four distinct kinds of cells, easily recognized the odontoblasts, Fig. 3, round, spindle shaped, and stellate cells. The odontoblasts form a continuous

Fig. 3.
Odontoblasts clinging to a fragment of dentine, showing their form. (Black.)

layer over the surface of the pulp, sometimes referred to as the membrane eboris. (See Fig. 4). This so-called membrane is composed of columnar cells lying close to each other, and sometimes present the appearance of being crowded out of shape. Each cell has four projections or processes.

1. The dentinal fibrils or fibers of Tomes.

2. Two lateral fibrils extending from the sides of each cell and uniting with adjoining cell to make a complete layer.

3. A process passing into the pulp tissue.

The fibers of Tomes are small projections which extend from the cell proper out through the dentinal tubules to the enamel and cementum. (See Figs. 5 and 5a and 6).

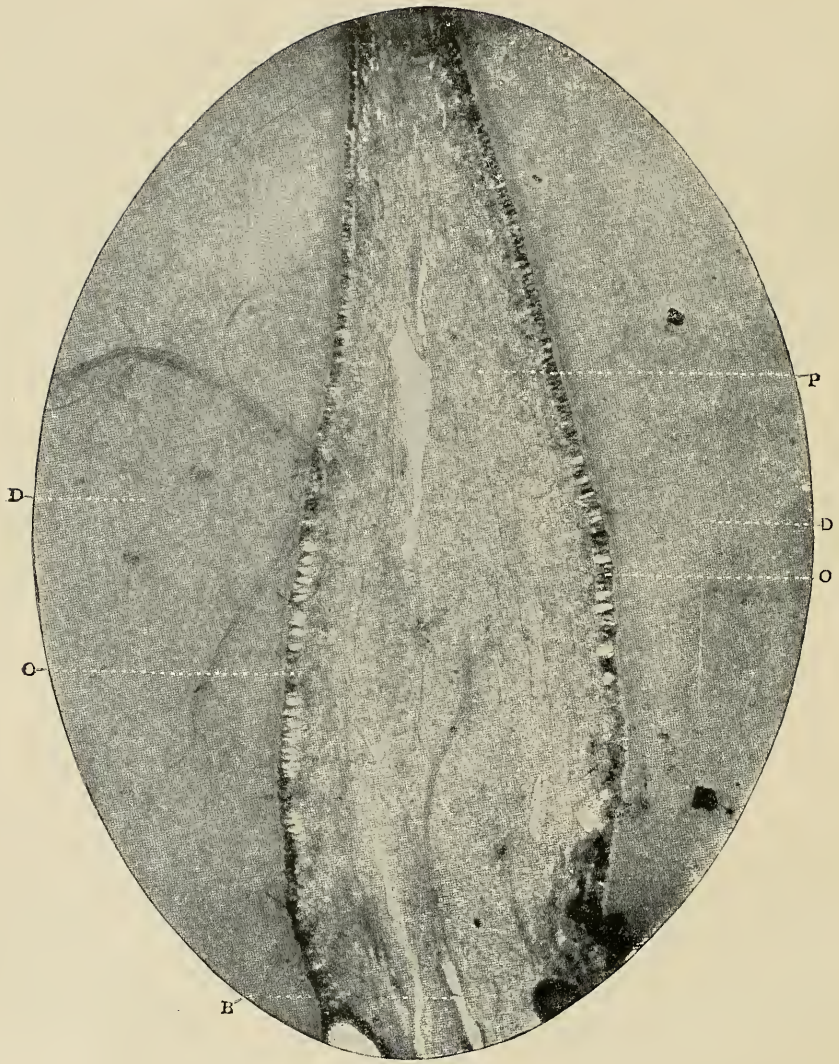

Fig. 4.
Longitudinal section pulp. O, odontoblasts; D, dentine; P, pulp. (Noyes.)

Fig. 5a.
Section of dentine from the root, cut in length of tubules. (Noyes.)

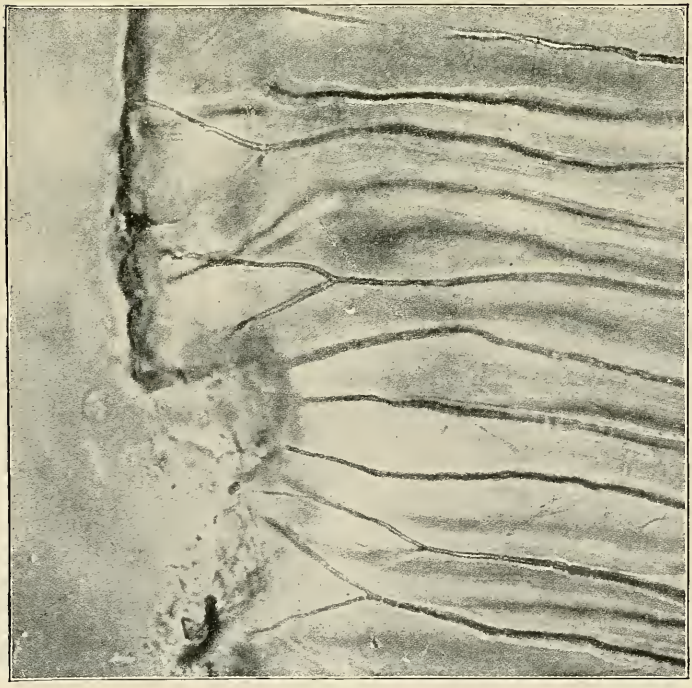

Fig. 5
Section of dentine in crown cut in length of tubules. (Noyes.)

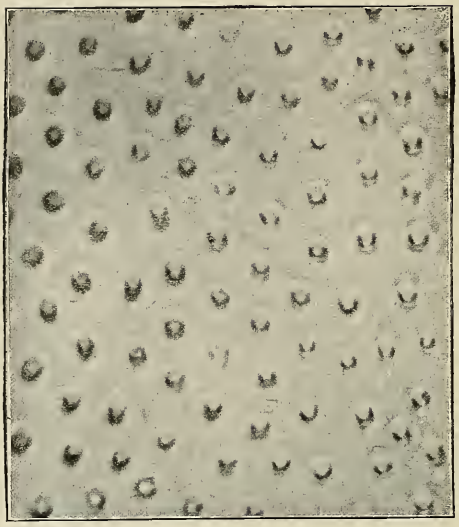

Fig. 6.
Section of dentine at right angles to tubules. (Noyes.)

The Functions of the Pulp.

The dental pulp performs two functions, viz., a vital and a sensory function. The vital function is the building of dentine, which is done through the odontoblastic layer. This function is most active while the papilla is large and dentine is forming, but after the tooth is once thoroughly formed this function seems to lie dormant unless some irritation excites the trophic centers, which may result in the formation of secondary dentine. The sensory function resembles that of the internal organs of the body—sensation of pains when irritated in any manner, but has not the sense of touch. This explains the difficulty we experience in locating the source of pain when it comes from a vital pulp. There is one other important point regarding the normal pulp which has a very direct bearing upon the pathology, and that is this—the pulp tissue completely fills the chamber and root canals.

Sensitive Dentine.

Normal dentine is not very sensitive, and the same is true of the pulp. The most generally accepted explanation regarding sensitiveness of the dentine is about as follows: The fibrils of Tomes, as we have before stated, are prolongations of the substance of the odontoblasts; they are a portion of the odontoblasts extending through the dentine to the dento-enamel junctions. These cells lie in direct physiological relation with the nerves of the pulp. (See Figs. 3 and 4.)

Other Cells.

There does not seem to be any regular arrangement for the other cells of the pulp. For the most part they may be said to be sparsely scattered throughout the tissue, assuming some regularity along the blood vessels. These cells are held in a gelatinous-like matrix with few fibers.

Blood Vessels.

The blood supply of the pulp is abundant; a number of arteries enter through the apical foramen, and extend occlusally through the central portion of the tissue, giving off many small capillaries (Fig. 7). The

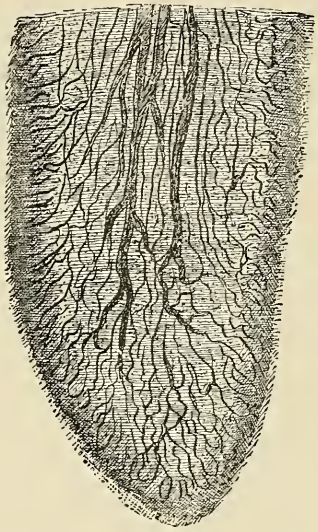

Fig. 7.
Blood supply in point of pulp. (Black.)

blood is collected into the veins and returns apically through the central portion of the pulp tissue, passing out through the apical openings. The blood supply does not always all come from the inferior and superior dental arteries, but sometimes a portion of it comes from the alveolar circulation, and occasionally there are lateral openings through the sides of the roots. An interesting specimen is now in the school museum. The thinness of the arterial walls is a peculiarity which we need to remember.

Nerve Supply.

Several large bundles of nerve fibers enter the pulp along with the blood supply and occlusally through the central portion, giving off many branches, which penetrate the entire tissue, even passing between the cells

of the odontoblastic layer. As yet no fibers have been seen to follow into the dentinal tubules.

Whenever these fibrils are irritated the sensation is carried directly to the sensorium through the central ganglia. The area of dentine that is most sensitive is at the termination of these fibrils—and in this particular they resemble other nerve endings. It is the finger tips that are most sensitive, not the deeper parts. This leads up to the consideration of hypersensitive dentine.

CHAPTER III.

Hypersensitive Dentine.

The Management of Sensitive Cases. Systemic Medication. Management of Sensitive Dentine. Obtundants. Thermal Sensitiveness.

The Management of Sensitive Cases.

Hypersensitive dentine is a term used to designate dentine which is unusually sensitive to filling operations; it is a subject that has attracted the best thought of the profession ever since filling operations began.

The proper preparation of cavities is often a very painful process; patients differ widely in this regard. A cavity that is unusually painful for one may scarcely be at all sensitive for another, although the cavity may be very similar and the process of preparation be identical. Why is this? Why do patients suffer so differently from similar operations? The reason may be one or all of three. First, the condition of the fibrils may be large and active to slight irritation, i. e., they may respond to the slightest irritation. Second, the nervous sensibilities may be more acute. A slight irritation may be magnified in its transmission to the brain. It may be true that this nerve of transmission is perverted in itself, magnifying the actual irritation before it gets to the center, and so the patient suffers increased pain from that cause. And the third, the fear or dread of the operation, may have induced an oversensibility in the pain centers of the brain, responding to the slightest irritation, etc. In preparing cavities in teeth containing living pulps we are usually causing pain, because we are actually working on live tissue capable of responding to the slightest irritation. The manner in which these dentinal fibrils respond to irritation has already been explained. Another element that enters into the cause of pain in these cases is the peculiar noise made by the instruments used in the work; particularly is this true of burs and stones in the engine. Patients frequently present themselves in a highly nervous, excited condition, due to meditating upon the fact that they have something to be done which they think will cause the ma degree of suffering beyond their power to endure. I might say that this is quite the usual attitude of mind that the patients present if they are coming to you for the first time. Not infrequently they faint upon sitting in the dental chair before you have done anything at all for them. This is an experience which I have had a number of times, and others, too, have had the same experience.

Our success in practice building will depend on our ability to decide what to do for these patients. If for a few minutes I seem to digress and preach a sort of sermon I hope you will excuse it. There are some things that I want to impress upon your minds now; they have been so profitable to me and so profitable to hundreds of others, and I think this is a good way to do it. The first thing, then, that we must do for these patients, presenting themselves in the condition that I have indicated, is that we must get their confidence. How is this done? I can't tell you. Each case perhaps itself inspires the operator with the knowledge of what to do; but I want to indicate to you along what line it comes. I am sure all of you have had cases that to work for was irksome, not because of the work itself, but because of the condition of the patient, or the attitude of the mind of the patient towards you. They have a sort of a notion that you are not going to do the work well, perhaps, or that you will hurt them needlessly, and you can never get them to have confidence in what you are going to do. When you get into practice here are some of the things you must consider. First, the personnel of the dentist. You must have a professional air about you. You want to recognize the fact that our calling is something more than trade; that it is a profession; there is a dignity about it, a professional air. Kind in manner and speech, and, as someone has said: "Keep your voice low." There is a whole lot in that. The dentist, of course, must be clean, quiet, cool-headed and in perfect poise. If you are nervous yourself about what you are going to do; if your mind is all uneasy and your hand is shaky and you are in dread of what you are going to undertake yourself, you can be sure that the patient will catch every bit of that and magnify it in himself or herself. I would like to cite you a little incident which came to my notice some years ago. One of my confrères in practice here in the city, a most thorough dentist and an excellent gentleman, came to me one day and said: "Doctor, I don't know what I am going to do; every patient that has come to me this week has fainted." I said: "How many hours a day are you working?" "About twelve." I said: "You look as though you were. How long since you had a vacation?" "I didn't get my vacation this year; I was so busy and had so much to do that I couldn't get away." "You had better take your vacation now; now is the time. Your patients are fainting because you impart this dread and fear to them yourself. You are all nerves; you are all unstrung; you can't do anything the way you want it done; you can't find your instruments, and when you do you drop them on the floor. You had better take a vacation." He took my advice, and when he came home from his vacation he didn't have any more people fainting in his chair. Then, you must give the patient your undivided attention. Don't hurry, take your time, and give the patient all your

attention. Then, of course, the dentist himself must be clean as to his morals. What we are shows in our faces and our demeanor in every way; we can't hide it; we can't debauch and conceal it. Then the next thing is the office itself. The office must be clean and tidy; there must be evidences that the patient's comfort is considered, and above all it must have a business-like air, i. e., it must appear to the patients when they come in that this is a place where people come to have dental work done. It is not a place to play in. It is a business office where everything is arranged for the comfort of your patients and to execute business. You don't know what an impression it makes upon patients for the first time if they find an air of business about your office in general. I once went into an office and saw in the reception room the gentleman's hunting boots that had been there from the season before, and a lot of traps pertaining to his hunting outfit laying there, covered with dirt and papers and circulars and things which will accumulate were thrown over this, and on every chair you could write your name in the dust. I went into his private office, and what did I see? On the bracket of his chair lay a forcep with an old tooth in it. That tooth, I am sure, had been extracted the day before, at least. The cuspidor was all covered with dried blood and sputum. I said to him: "How is business?" He said: "Business is bum." That gentleman tried to practice in Chicago for upward of fifteen years, and he has gone to a little town of one thousand inhabitants now. Not that he was not a good dentist, for he was, but the inattention to all these details disgusted everybody. Then, have an absence of disagreeable odors about the office. How often you have patients tell you that the odor about a dentist's office is the hardest thing for them to bear. You do not need anything of the kind; have the cuspidor clean. And do not have any instruments in sight; that is a hobby of mine. When I first began practice I thought a pretty good stock in trade would be to have a nice array of nickel plated instruments in sight. So I started out with that idea, and patients would look in the direction of these instruments, especially the extracting forceps, and wish they hadn't come. I never have any instruments in sight now; I mean by that, when the patients comes into my operating room to have their teeth examined they sit in the chair, and there is not an instrument in sight. I have my case convenient and my instruments so arranged that I can put my hand on any instrument I want immediately. Then after they are used the young lady takes them and sterilizes and sharpens them, and puts them back where they were before. This is a thing that I have no special patent on; others do it the same as I do. Then you want your instruments clean. Nothing will disgust a patient so quickly as for you to dismiss one patient and invite another one in with the dirty instru-

ments you have been using lying on your bracket. Perhaps people who do not think of these things do not care, but the class of patients that you want do care. The world over the laity understand about infection; they understand about the danger of carrying disease from one to another on instruments, and you will fool yourself if you think they don't. I have had any number of patients come to me for the reason that their dentist seemed to use instruments on one after another without sterilizing. In the first place it isn't correct practice; if you should infect a patient in that way you would be liable for malpractice; and in the second place, it is absolutely false business principles. Then attend to the details for the patient's comfort. One of those little details is this, and will serve to make clear my meaning: patients often come with their lips chapped, and you should have a little cold cream or something of the kind, that you can smear on the lip to protect it. Let the patients have an idea that you are thinking a little about their comfort. Then assure your patient that your aim is to work with as little pain as possible, and do operations well, that you will not hurt needlessly. I say to patients: "I will tell you when I am going to hurt, and so you need not be expecting it until I tell you." That helps a whole lot. And then they don't worry about it. And get your patients calm and relaxed before you attempt to do anything. Make them let go of their nervous tension. You will find patients when they come for the first time will usually grip hold of the arm of the chair, put their feet down against the footrest and stiffen themselves right up—just the opposite to what you want them to do. The first thing to do is to explain to them the philosophy of the action of their nervous system regarding pain, which can be done in half a dozen sentences, and get patients to release their hold on the chair, drop their hands at their side or into their lap and relax themselves entirely; tell them to take in a few full breaths, quiet and restful; lower your voice and talk to them in that way, and tell them to just forget themselves and let their mind be relaxed. I do not know anything about hypnotism, but if this is hypnotism that is the thing you want to practice; it will bring you success. I think I used to suffer more than anybody else having my teeth attended to; dentists always do. When you get into practice and have 25 or 30 dentists on your regular list you will find that they always think they are the ones who suffer the most. I was sure I did. I used to have a terrible time. I got to thinking about this thing, and I concluded I would practice on myself some of the things I was preaching. I go to my dentist and sit down in the chair, and in a few minutes I get myself so relaxed that scarcely any operation in the mouth causes me any pain whatever. Not that I am not just as sensitive, but because I am able to relax my entire system in such a way that I suffer less than others do. That is the

kind of thing I want you to get in the habit of doing. You will be astonished to find how it will aid you. Then after you have done this undertake something easy the first sitting and gradually work towards the more difficult and severe. When your patient trusts you implicitly you have won a lasting friend and a most profitable patient. The fact of the matter is, if you will just take time to do this thing the first time the patient comes, you will have no trouble in keeping that patient as long as you are in practice. They will come to you under all circumstances. It is the first visit that bothers all of us. I have patients, and so has every practitioner, the first time they came to me was almost killing on myself to do anything for them. Every move you make their hands will go up and catch your hand. "Oh, dear, I really don't think I can stand this; you will have to let me rest awhile," and all that sort of thing, and you put in an hour or two in doing something that you could do in five minutes.

But when they get confidence in what you are going to do, you will not have any trouble. I have people with whom I have had just such experience, who come to me now without the least dread, and it is a positive pleasure to work for them. I remember one case in particular about eight years ago. I was associated in my office at that time with a very estimable gentleman. This patient made so much fuss that it disturbed his patient and himself and everyone in the office, over work that was not painful in itself, and I pretty near killed myself trying to do decent work for her the first few visits.

Finally my associate came to me and said: "There is no use talking, you will have to let that patient go or she will drive everybody out of the office." I was quite inclined to adopt his notion of it; but I finally got through with that series of operations, and I have had no trouble with that patient since. She comes to me regularly and has sent me dozens of others. She came to me from another dentist because his operations were not successful; she was losing fillings all the time. Why? Not because he wasn't a good dentist, but because he couldn't get that woman into condition where he could work for her. If you cannot get a patient's confidence let him go. You can't afford to do otherwise. Working for people who are set against everything you are going to do is absolute folly, because you are killing yourself, you are killing the patient, you are doing poor work; you can't help yourself, and it will add nothing to your credit, and in building up a practice you do not care so much for cases that just happen in to have something done. What you want are families that are going to come to you year after year, that are going to say a good word for you among their neighbors and friends, and because you are just starting in practice and need every dollar you can get will

lead you to undertake things, just for the sake of that income which you cannot properly do. Such a procedure is absolutely foolish business policy. If you dismiss that patient with a distinct understanding, say to him: "You have no confidence in what I am trying to do for you; you have set yourself against what I am trying to do, and under those conditions I cannot do you good work; I am unwilling to do what I know to be poor work, and you will have to go to someone else." That will often bring them to their senses and they will appreciate what you are trying to do. Make a practice of studying each patient; no two are exactly alike, and what I have said will not apply to each case, but will serve to indicate the method of procedure. In managing children in order to have them bear things that are painful requires a good deal of tact. And your success in managing these little folks has much to do with building up a practice, for it is astonishing how rapidly little folks grow up to be big folks.

Systemic Medication.

For the purpose of allaying this nervous irritability we frequently need to call to our aid some systemic remedies. Hypnotic anodynes, agents that have the power of allaying sensibilities of the nerve centers, or the peripheral terminations, are those of most value to us. In this class belongs opium and its various preparations and most important alkaloids, chloral, trional, sulphonal, bromide of potassium. Morphine is one of the alkaloids of opium, and the one most used. For purposes of alleviating pain it is without a rival. It is given in doses of 1/8 grain an hour before the operation, and another fifteen minutes before. I never tell my patients what they are taking when they take morphine, because so many have a prejudice against it. When they come in for examination and you recognize that you are going to have trouble, you can give them one of these little tablets, and tell them to take it an hour before they come the next time, and when they come to the office you give them another one.

Dover powder is the form of opium frequently given for allaying painful conditions, especially about the peridental membrane, in 5 gr. doses.

Codeine, another alkaloid, is frequently used; it is much less powerful than morphine.

Chloral is often of value, especially for the purpose of allaying nervousness where there is little pain to be endured. In large doses chloral produces sleep quite like natural sleep, from which the patient can readily be aroused. If operations are painful it is not so valuable as the opium preparations, but I have had very excellent results in doses of from 5 to 20 grains simply for the purpose of quieting the patient.

Trional I have tried with success in many cases in doses of from 15 to 30 grains, also sulphonal, tetronal and hydronal. Fluid extract of Jamaica dogwood is said to be of value, but personally I have never seen its value. I have frequently received surprising results from the use of bromide of potassium in 10 grain doses. Next to opium I rely on this. I have never received any assistance from the coal tar antipyretics. You will find in the literature men recommending these for the purpose of allaying nervous irritation, but personally I have received no benefit from them.

Management of Sensitive Dentine.

Locally, i. e., to the cavity itself. Have the cavity perfectly dry. Never attempt to excavate a sensitive cavity without the rubber dam on. Use warm air and alcohol to assist you. Use only sharp burs, and if you have teeth to be filled just try the experiment of having the operator use a dull bur and a sharp one, and note the difference in the amount of pain they induce. Have your excavators sharp, avoid overheating and make no false moves, but make each stroke count. When you are not going to hurt say so, and when you are tell the patient: "This will hurt a little bit; I will be careful, and it will be only for a moment."

Obtundants.

For purposes of obtunding the sensitiveness in the cavity many substances have been used. Once upon a time men used arsenous acid, sealing it for twelve hours, but all those pulps died. Then came the use of chloride of zinc, which is still used by applying the crystals directly to the cavity and allowing to liquefy in the cavity, i. e., allowing the crystals to absorb sufficient moisture to become liquid, leaving it for fifteen minutes or so. This frequently assists greatly. You must not use chloride of zinc when your pulpal wall is very thin, or too near the pulp. The next agent which was used is nitrate of silver. This, perhaps, is as efficient as any agent we have, but because it discolors the teeth it cannot be used in any anterior teeth, and I never like to use it in a cavity at all, except in children's teeth, of which I will speak later. But around sensitive margins in the posterior teeth, where no actual decay occurs, it is of value. The method that I employ of using it is this: I make my solution fresh each time, making a saturate solution of nitrate of silver in sterilized water. Then I take an orangewood or rosewood stick, whittle it flat like a spatula, then dry the surface to which it is to be applied, dip the point of this stick in the solution and rub it back and forth over the sensitive part, doing this four or five times, and avoid getting it on the soft tissue. I find that to be a very efficacious method. Others crystallize this nitrate of silver on the point of a platinum wire, and then allow the moisture to remain on the surface, rubbing this point over the moist surface. Others

use it by making their saturate solution and then cutting little squares of asbestos or blotting paper. This blotting paper I had prepared for the infirmary as far back as 1892, and I am surprised to have someone bring it forward as an original notion of his. When you wish to use it, allow the surface to be moist, and take up this little piece of paper and rub it back and forth over the surface. That is a very nice way to have it for purposes of applying it to the soft tissue where you want to burn out a mucous patch, little ulcers that occur on the tongue and around inside the lip that are painful. But I have got in the habit of using it in the method spoken of first. Others use a silver wire and dip it into nitric acid, forming their nitrate of silver direct.

Now, care must be taken in using these preparations not to get them over the soft tissue as it will burn the tissue wherever it touches. If accidents do occur with this, what is the thing to do? Use salt and water, forming a comparatively insoluble chloride of silver, and you have corrected the harm the quickest way you can. When the surfaces are badly discolored from its use, and for any reason you wish to bleach it, use a solution of cyanide of potash, or another way is to paint the discolored surface several times with the tincture of iodine, rubbing it back and forth until the iodine has actually cut the stain; then bleach your iodine with ammonia.

Absolute alcohol is a valuable obtundant. It should be applied directly to the cavity with a fine spray and continued for a few minutes. The benefits derived are due to the extreme dryness caused. A mixture of ether, chloroform and alcohol is valuable used in a similar manner. Ether, chloroform, alcohol, menthol in equal parts has been suggested as a valuable obtundant. This mixture should be used in an especially made compressed air atomizer. A very fine spray is what is needed and should be directed into the cavity while excavating or burring. Rhigoline, a light petroleum ether, has been recommended. Ethel chloride,

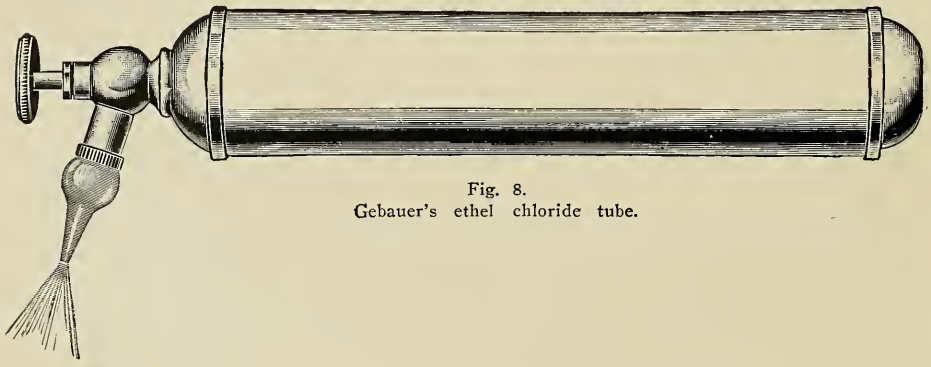

Fig. 8.
Gebauer's ethel chloride tube.

which is sold under many names by different firms, is a valuable remedy. It comes to us in a special container made of thin glass or metal, with a stop screw so arranged that it can readily be opened when the heat of the hand will cause a fine spray to spurt out; this spray should be directed into the cavity a few moments before beginning work and repeated frequently while excavating. Fig. 8. All of these ether mixtures obtund the sensitive dentine by their excessive drying action and also by the extreme cold they cause, and therefore some care must be exercised in order to avoid pain while using and death of pulp as a final result.

Carbolic acid applied warm to the cavity will often prove helpful.

Cocain, used in connection with ether, is of value, and also used under pressure in a similar manner is applied for immediate extirpation of pulps. That cocain used under pressure will obtund sensitive dentine there can be no doubt, but the after effect upon the pulp is a matter still to be determined.

Cocain citrate sealed in the cavity for 24 hours sometimes proves helpful. Many operators inject one per cent. cocain hydrochlorate solution into the gum tissue the same as for tooth extraction, which will often prove helpful. Recently it has been suggested that *nitrous oxide* be administered. The Hurd, Clark and Tetter inhalers have been made for the purpose. The plan is to have the gas given by way of the nose and not to completely anesthetize the patient; but just enough is given to produce semi-consciousness, which condition can be maintained for several minutes, during which time the excavation is completed. The author has never had much success with this method. The difficulty of preventing the patient taking air through the mouth and maintaining dryness while working, added to the dislike patients have for taking the gas seems to indicate that the plan will never be generally adopted.

Hemicranin dissolved in nitrous ether sealed in the cavity for a few minutes will often prove helpful.

In concluding this subject I wish to emphasize the importance of working with *precision* and *decision,* using sharp instruments, and when possible cutting the fibrils a little way from their terminations particularly in labial and buccal cavities, absolute dryness is essential to success.

When all has been said the fact remains that some cases present that will not yield to any measures suggested, and it is my teaching not to attempt the impossible. If permanent operations cannot be made without too much suffering then by all means do temporary work and have it so understood. When cavities are filled with oxyphosphate

cement for a year or so the fibrils lose their sensitiveness, when permanent fillings can more easily be made.

Thermal Sensitiveness.

The tooth pulp is peculiarly sensitive to thermal changes. Everyone has had the unpleasant experience of allowing ice water to come suddenly in contact with the teeth. The sensation is always one of pain more or less severe according to the condition of the pulp. Normally the pulp responds in this way to excessive changes of temperature and cannot differentiate between heat and cold. Within reasonable bounds this is normal, but under certain conditions it becomes hypersensitive to these changes—the slightest elevation or reduction of temperature produces pain, and, while this is unpleasant to the patient, it is sometimes of diagnostic aid to us, to which I shall refer in another chapter. When such conditions present you may feel very certain that changes are going on in the pulp itself. There is present a pathological condition and at least the beginning of hyperemia of the pulp, which is the subject of another chapter. All I wish to say here in this connection is that there is an injury to the blood vessels of the pulp accomplished by traumatic or chemical irritation through the fibrils and odontoblastic cells as a rule, but may be brought on by a variety of things—the clearest statement I find on this point is in Dr. Black's lectures, page 216, which is as follows: "Thermal sensitiveness is liable to be aroused in many different ways. I have suffered from it myself occasionally in my incisor teeth from its being aroused from heat of a cigar in smoking, so much so that I had to either cease smoking or protect the teeth. It may be caused suddenly by an extraordinary exposure to cold, as ice water. It may be caused suddenly by exposure to hot drinks, and the dentist may develop it suddenly by the heat of the disc in finishing a filling or the heat of a bur in excavating, in any tooth that has a living pulp. Often the thermal sensitiveness is aroused during the progress of decay, especially when the decay has reached the neighborhood of the pulp of the tooth, and the patient will have paroxysms of pain continuing longer. This continuation of the paroxysms of pain marks the severity of the case, and finally, if it continues to grow worse, the patient will have pain when lying down, will have pain at night; the difference in blood pressure between the horizontal position and the upright position will be sufficient to determine a condition of pain. They will become sensitive as that. I have seen cases in which throwing of a stream of water on the tooth three degrees off either way from the normal temperature of the body would induce excruciating pain. In the management of cases it is the utmost importance that we recognize what may occur, and due caution in regard

to running disks dry, or even in running them wet we may sometimes produce too much heat, or running burs too long in excavating, or any of these things that are calculated to produce heat which may suddenly precipitate a condition of hyperemia of the pulp or thermal sensitiveness.

What will we do for it? There is only one thing to do, and that is to protect the case as absolutely as possible from thermal changes until it recovers. That may be done in various ways. In some of the worst cases I have put caps of gutta-percha over the teeth involved, covering them in completely, particularly with persons who must be out in the cold air, and where I could not otherwise induce persons to protect them from thermal changes. The patient himself, or herself, may protect the teeth from thermal changes; they may avoid cold or hot drinks; they may avoid cold or hot foods; they may avoid breathing from the mouth, and in this way protect the teeth, and it is very much the best way to protect them from thermal changes. If we put gutta-percha caps over the teeth they will be very annoying, and it is often difficult to induce patients to wear them.

Cases of very severe thermal sensitiveness will get well if properly protected, generally promptly, within a week or ten days. Sometimes, however, it may require more time, and wherever we find them developed to any extraordinary degree, so as to be very annoying, we should desist from all operations upon that tooth except those calculated to mitigate this condition. If it has occurred from a cavity of decay it is best to remove all decayed dentin completely, so as to remove the irritation caused by the irritants in the decaying mass. When the cavity is prepared do not make a filling, but make a temporary filling of gutta-percha, and be sure you make a tight filling. Have the walls dry first, and moisten them with eucalyptol, so as to have your gutta-percha adhere and make your filling tight. This is the best treatment, for gutta-percha is the best non-conductor we have with which to make these temporary fillings. A gold filling at that time would be the worst thing that could be done. Then await the cure of this condition before making any other operations upon that tooth, and if it is severe avoid any operations whatever in the mouth until that tooth shall have recovered, or at least any operations that are not absolutely necessary at the time.

Now, this condition often ends in death of the pulp from strangulation or infarction. Today the tooth may be extremely sensitive to thermal changes; tomorrow the pulp may be dead, and this sensitiveness may have disappeared completely. The sudden disappearance of this thermal sensitiveness marks very certainly the death of the pulp, and when the pulp of a tooth has died under these conditions it is of extra

importance that we get the pulp out as quickly as possible. When recovery is complete in these cases it is usually by the pulp receding from the point of irritation and depositing through the odontoblasts a layer or layers of secondary dentine.

CHAPTER IV.

Constructive Diseases of the Pulp.

Secondary Dentine and Pulp Nodules. Causation. Pulp Nodules. Symptoms. Calcific Degeneration of the Pulp.

Secondary Dentine and Pulp Nodules.

By the term secondary dentine is meant dentine formed about the walls of the pulp chamber abnormally. Pulp nodules is a term used to define irregular masses of calcic material occurring within the pulp tissue. These appear to be slightly different phases of the same process. The pulp seems to throw out a layer or layers of dentine or bony substance resembling it, as a means of protecting itself from the irritation of encroaching caries, erosions, abrasions, and from the thermal irritation as a result of large metallic, especially gold fillings. In the majority of cases where gold fillings are placed teeth are more or less sensitive for some weeks, and only passes away as new tooth material is deposited in the tubuli over the point of pulp exposed to such irritation. With advancing years pulp chambers become smaller, made so by the continuous deposit of dentine; the lumen of the tubules lessens. This process goes on until the pulp is almost obliterated in extreme old age.

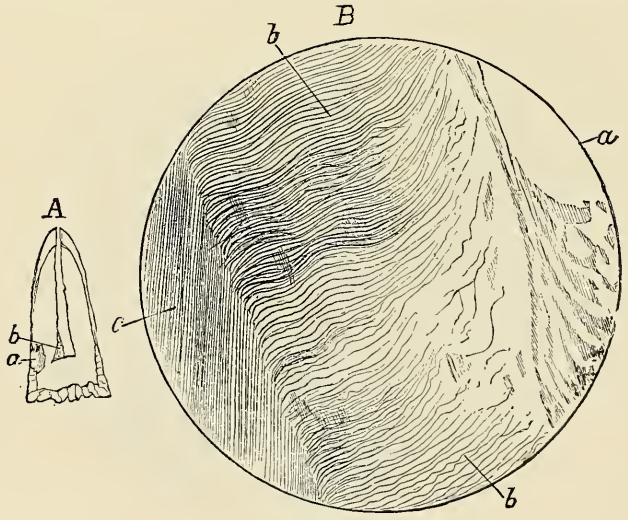

Fig. 9.

Deposits of secondary dentine. *A*, section of an incisor showing caries at *a* and secondary dentine at *b; B*, section of secondary dentine; *a*, pulp chamber; *b*, *b*, secondary dentine; *c*, primary dentine—notice directions of tubules in each. (Black.)

Within certain limits the formation of secondary dentine is purely a physiological process, and seems to be a part of nature's scheme to protect the pulp, and is deposited by the odontoblasts.

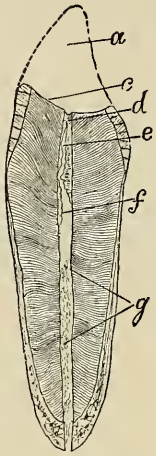

Fig. 10.

Secondary dentine filling the pulp chamber of an abraded cuspid. *A*, part abraded; *c*, the abraded surface; *d*, secondary dentine; *e*, a slender point of pulp; *f*, deposits on wall of canal; *g*, deposits in pulp tissue. (Black.)

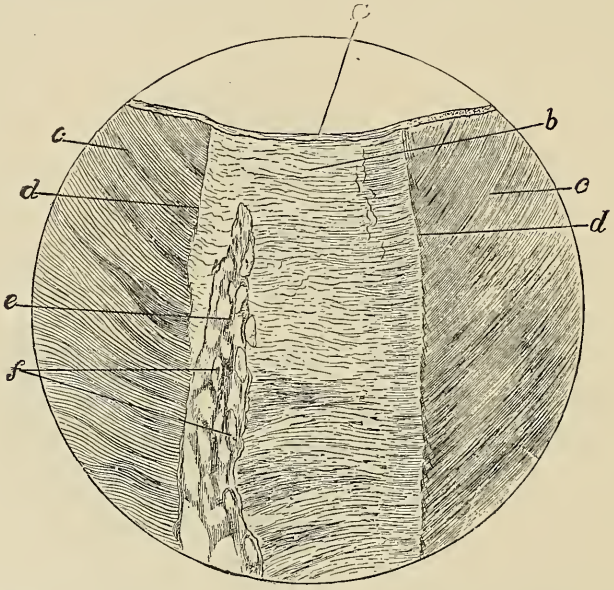

Fig. 11.

The same as Fig. 10—magnified to show difference in primary and secondary dentine. D. normal dentine; *b*, secondary; *e*, remains of pulp; *f*, small oval deposits in pulp.

Secondary dentine differs anatomically from normal dentine, from which it can usually be distinguished by its lessened translucency and changed direction of the tubules; indeed, the microscope will show fewer tubules in the secondary than in the normal dentine, and the line of demarcation can easily be seen. (See Fig. 9.)

The extent of these deposits is important to note. Some observers think it never progresses so far as to completely fill the chamber and root canals; others have cited cases where the entire canal seemed to be filled. I have had several cases where the upper two-thirds were completely filled as a result of wearing down the teeth. They were abraded a considerable distance below the covering of the normal chambers. (See Figs. 10 and 11.)

In the molar teeth these deposits occur most peculiarly. In some instances simply a small amount immediately under the carious cavity only; and in other cases the deposits will not only be at that point but on the floor of the pulp chamber as well when the normal grooves in the floor will be obliterated. In other cases the deposits will occur on opposite walls and progress until the chamber proper is completely obliterated (Fig. 12). In still other cases it will deposit in well rounded tumor-like masses held to the wall by a narrow pedicle.

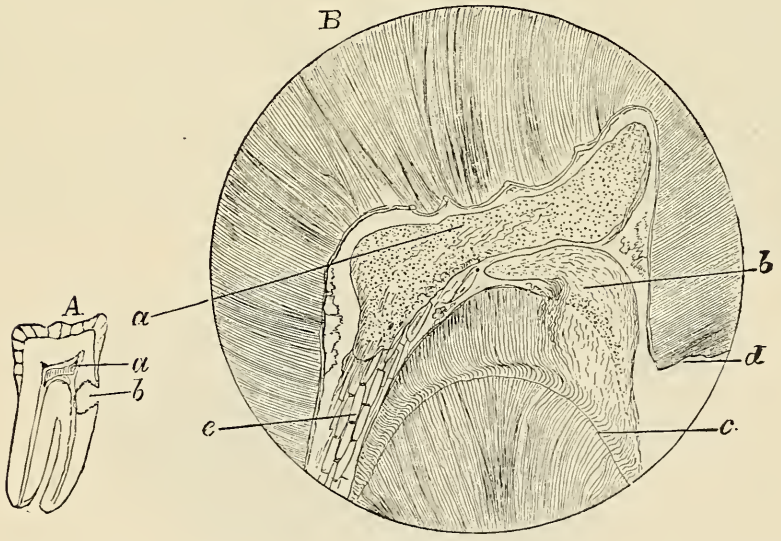

Fig. 12.

A, outline of lower molar with cavity at *b*; at *a* is seen a large mass of secondary deposits: *B—a*, the granular mass of secondary deposits; *b*, a little different form of deposits, and *e*, cylindrical form of deposits; *c*, growth from floor quite regular in form; *d*, outline of cavity. (Black.)

I have seen some cases of one rooted teeth where the deposit occurred on the walls of the root canal, producing a stricture, as it were, of the pulp.

Causation.

Very little can be said regarding the cause of secondary dentine; much has been written, most of which is conjecture. Reasoning from the cases we know about it would seem to be due to some mild form of irritation from without, to which I have previously alluded, or to some trophic nerve disturbances. In all instances it seems clear that there must be a constantly recurring hyperemia of a very mild type, for in all of these cases of purely secondary dentine we get no history of pain of any marked degree, and in most instances no pain whatever. The clinical importance of these cases relates to the fact that sooner or later the pulp is lost, and in case the pulp has to be removed for any cause it makes the accomplishment of that end more difficult. This point will be referred to again in the chapter on capping pulps, and also in the chapter on the management of root canals.

Pulp Nodules.

As before stated, they are irregular masses of bone-like substances occurring within the pulp itself. They resemble secondary dentine in chemical and physical construction, but differs from it in anatomical characteristics. They seem to more nearly resemble bone. They are not built by the odontoblasts, but the exact method of their formation seems to be in doubt. Dr. Black, who seems to have done most work in studying out these formations, describes three distinct forms.

1. Deposits of calcoglobulin, in form resembling the other two, but soft and always found in the inflamed portion, usually just beneath the odontoblasts. (See Fig. 13.) Some think these are masses from which nodules are formed.

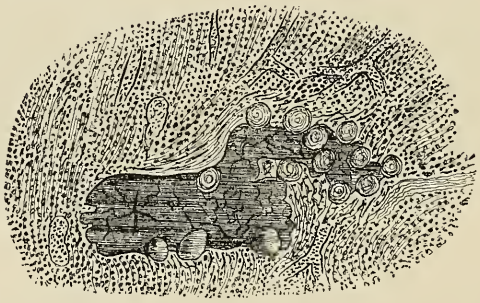

Fig. 13.
Deposits of calcoglobulin within the pulp tissue.

2. Calcospherites—hard round bodies differing in size, but formed in layers like the onion. (See Fig. 14.) These seem to be formed in any portion of the pulp, and are usually mixed in among the nodules and sometimes are contained within the nodule itself.

Fig. 14.
Pulp nodules, tooth of a whale. (Barrett.)

3. Pulp nodules, the hard irregular masses above referred to and illustrated in Fig. 15, occur throughout the pulp tissue, but more abundantly in the crown portion, and we are sometimes astonished to find such large numbers of them within a single pulp. One case in my practice I succeeded in removing 66 distinct nodules, and yet the tooth had given very little trouble to the patient. Dr. Black is of the opinion that they originated within the veins of the pulp and are more abundant in middle age. They seem to occur in perfectly sound and otherwise healthy teeth.

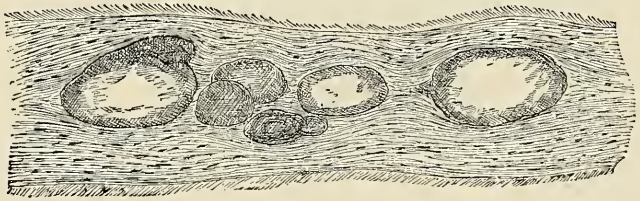

Fig. 15.
Pulp nodules in the central portion of pulp. (Black.)

Symptoms.

Pulp nodules may exist in great numbers and give rise to no discomfort, as I have often seen in extracted teeth, which have given no

history of pain, and also in cases where pulps were destroyed for bridge abutments.

Then again the pulp of a tooth may be the seat of the most excruciating pain without the least external evidence as to cause, and when the pulp is destroyed it is found to be filled with very small nodules. Reflex pain is very commonly associated with these deposits, and pulps containing them become exceedingly painful to the mildest irritation. Persistent neuralgia with the pain deflected into some distant organ or part of the head is very commonly found to be associated with these deposits. Indeed, after 15 years' experience in handling these cases I have come to the conclusion that when these neuralgic pains persist about the face and jaws and also the ears, and I cannot find any external signs about the teeth as the probable cause, the cause is the formation of these nodules or other deposits within the pulp chamber of some tooth which can only be located by using all tests known, and sometimes by cutting into the tooth and devitalizing can the fact be found; although hypersensitiveness to thermal changes or little or no response will often aid in locating the offending tooth. Treatment must always be devitalization and removal of pulp involved, which is by no means an easy matter in many cases, requiring patience, perseverance and weeks of time. This will again be alluded to in the chapter on devitalization.

Calcific Degeneration of the Pulp.

Calcification of the tissues of the pulp is another problem presented. This is a condition which the pulp itself, especially the fibrous tissue, becomes calcified, resembling calcifications occurring elsewhere in the body. It seems to follow certain degenerative changes in the pulp tissue itself, usually low chronic inflammation. The morbid anatomy presents some marked differences from that seen in the nodular and dentine calcifications. In this variety the pulp tissue itself seems to be converted into hard bony substance, and in the root portion assumes a cylindrical form apparently about the fibers. (See Figs. 16 and 17.) In some cases this calcification may go on until the whole central portion of the pulp is involved, extending from the crown chamber to nearly the apical foramen. I have such a specimen now where I succeeded in removing a bony cone from an upper cuspid which is nearly as long as the root canal and chamber, and is surrounded by a thin layer of pulp tissue. In this case there was no symptomatology. Indeed, in any cases there does not seem to be any differentiating symptoms. In concluding this chapter I wish to cite a case that came under my observation, as follows:

A gentleman about 48 years of age came to the office complaining of severe pain in the right side of his face, a sort of paroxysmal pain.

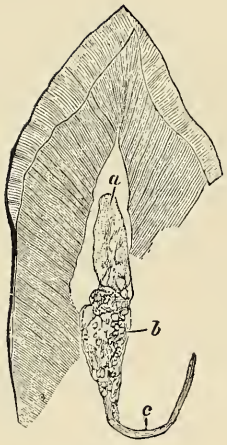

Fig. 16.
A, entire pulp; *b*, pulp nodules; *c*, pulp without nodules. (Schlenker.)

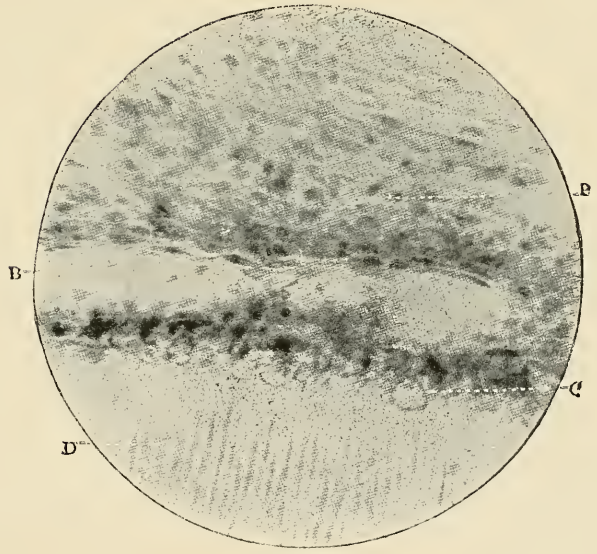

Fig. 17.
Acute inflammation of the pulp. D, dentine; P, pulp; B, enlarged capillary; C, calcoglobular mass. (Hopewell Smith.)

Examining his teeth, I could not find any cause for trouble excepting that the occlusal surfaces of the molar teeth were abraded mechanically, the cusps were worn down. The application of the usual means of detecting hyperemia failed to detect any. I used in his case electricity, together

with a liniment and massage to his face, and it seemed to relieve him somewhat for a time. By and by these pains would come with increasing intensity, so that the muscles of the face would undergo spasmodic contraction beyond his control entirely. Well, he was an individual who wouldn't bear much work with his teeth. He was one of those individuals who didn't believe much in having much done with his teeth anyway, and consequently had neglected them until he had lost a number of his molars. He decided on his own hook when we couldn't find the cause of the trouble, that he would have a certain one out, and he went and had a lower second molar extracted. He brought the tooth back to me and I cracked it open. The contents of the pulp-chamber and the three canals were completely calcified, so that I removed it all in one piece, a thing which occurred in successive teeth of his until five teeth went in the same way. I don't know what the cause was. It didn't scarcely seem probable that the mechanical attrition was the cause. I rather think it was something of a systemic nature that had to do with it, but I don't know.

Hypertrophy of pulp will be treated under inflammation of pulp.

CHAPTER V.

Destructive Changes in the Pulp.

Hyperemia. Causes of Active Hyperemia. Causes of Passive Hyperemia. Causes of Hyperemia of the Dental Pulp. Painful Process. Discoloration. Symptomology. Treatment.

Hyperemia.

It does not seem to me desirable that I should here go into a lengthy explanation of the hyperemic process other than as seen in the tooth pulp. Your studies in general pathology have made you familiar with this general subject, and yet I think it wise to present concisely the main facts. For further consideration you are referred to general text books on the subject of pathology and surgery. Hyperemia is a condition in which there is an increased amount of blood in the part. When due to increased arterial flow of blood it is called *active hyperemia,* and when due to an obstruction in the flow of blood away from the part, we call it *passive hyperemia.*

Causes of Active Hyperemia.

Active hyperemia may be purely a physiological process; unusual exercise of any organ calling for increased nutrition through the circulation will cause a physiological hyperemia. That is the method by which nutrition is supplied. The blood supply of the tissues and organs of the body is under the direct control of the vaso-motor system of nerves; when any part is in need of increased nutrition this fact is, as it were, telegraphed to the centers, and at once the arteries and capillaries relax, and an increased amount of blood passes through, and so long as the normal physiological function is maintained in the part we have purely a physiological hyperemia; but if carried beyond this, and we have a considerable amount of blood with widely distended vessels, injured walls, etc., we have a pathological hyperemia, and the beginning of inflammation. Active hyperemia may be caused also by disease of the heart, e. g., over activity of the heart or disease of the arterial walls, they being too relax, etc.

Causes of Passive Hyperemia.

Active hyperemia is liable to become passive. After a time, from overwork or injury the veins fail to carry away the blood as fast as it is supplied; there is then a slowing of blood movement in the part. The vessels' walls lose their tone and the blood collects and become stagnant. I mean by stagnant that there is not sufficient oxygen in it; then we have

passive hyperemia. We have passive hyperemia from still another cause, namely infarction, when from any reason some semi-solid substance, a blood clot, calcarious matter, an embolus, or what not, breaks away from its original moorings and floats into the blood stream until it lodges in a small artery, which in turn collects other blood cells to itself, resulting in complete or partial stoppage of the vessel. Blood continues to flow in increasing quantities to the part; little or none passes away; then we have truly a pathological condition, which soon results in serious inflammation. The amount of blood stagnant in the part will depend somewhat on the number of anastomosing branches in the exact locality. Infarction can be artificially produced. Let me say that the method used for demonstrating that is to take some of the lower animals, more particularly the frog. Conheim, for example, took little balls of wax, opened the aorta and dropped these balls of wax into it. Then as they passed along the circulation he lifted up the tongue, and could see the balls of wax stopping in the arterioles under the tongue.

Illustrating the fact that when these things that I have spoken of as an embolus, a little blood clot, or a little calcarious matter, or anything of the kind that has formed and becomes attached somewhere, breaks loose and floats down the stream, and when it gets to where it can't go any farther it plugs it up, and as a result we have blood flowing to the part in an increased quantity, none going away, and we have as a result, infarction and passive hyperemia. If we watch the tissues under the microscope during the process and progress of hyperemia what do we see? The most common experiment is to take the web of a frog's foot, stretch it tightly across the field of the microscope and notice the blood flowing; you can see it readily; then irritate the part so as to injure the tissue. First, you will notice a slight contraction, followed immediately by distention of the vessels. The white corpuscles begin to hover around the injured tissue; the vessels continue to distend; by and by the serum of the blood begins to escape through the vessel wall. Next, the red corpuscles work their way out into the tissue in increasing numbers. The part becomes swollen and red, due to the increased number of red blood cells in the tissue, and finally we have complete infarction and stasis. This is the thing that often occurs in the dental pulp, and it is exactly at this point that we have the beginning of inflammation.

Causes of Hyperemia of the Dental Pulp.

First, and most common, perhaps, is a simple irritation of the dental fibrils. You are all familiar with the progress of decay into the cavity. A small break occurs through the enamel, and then underneath the enamel a great hole is burrowed out, always keeping conical in shape. In

the progress of this decay these micro-organisms actually work into the dentinal tubules and there perform their function, i. e., the function of growth and reproduction—the function of nutrition. They take their nutrition first by producing something to act as a solvent of the things to be taken into the system, in the process of which they evolve their irritant waste product, which continues to irritate these fibers, and in turn the odontoblasts are irritated, and they in turn affect the pulp itself, which calls for increased nutrition in the part; the poisons formed from solution of their dead bodies act in a similar manner. It is a very important thing for us to remember that these micro-organisms produce irritation by all of these ways. This irritation may occur through abraded or eroded surfaces by the influence of the irritant action of substances such as acids, heat, cold or attrition.

We have another class of cases where there is no decay. Teeth apparently sound suddenly become sensitive to thermal changes. It may be caused by continued contact with heat, such as the cigar or pipe, or something of that sort, or it may be from some systemic disturbance which dilates the arteries—hyperactivity of the vaso-dilator or vaso-constrictor in the vessel wall, a thrombus in the circulation, a shock of some kind—all of which may give rise to hyperemia of the pulp, and not infrequently, death. Sometimes this occurs from overheating with burs and stones, polishing strips and disks, as I have stated. I think I ought to emphasize that. It is so easy to cause hyperemia of the pulp by excessive heating from our instruments. More particularly do I think that students are likely to cause this by the use of their strips, taking strips and drawing them from one end to the other between the teeth, filing down their fillings, in that way causing excessive heating of the part, a thing to be avoided. When the pulp is irritated from such a cause what occurs? First, if the irritant be of mild nature we have a physiological hyperemia, increased nutrition carried on and a protection thrown out and a drawing back of the pulp and new dentin formed, usually over the spot of irritation, i. e., irritation to the odontoblastic layer and then irritation to the pulp itself; and the pulp will gradually recede from this irritant, and throw out through the medium of the odontoblasts a second deposit of dentin, thereby protecting the pulp tissue itself from the irritant. That is nature's method of protecting the pulp from the irritant without. Sometimes this physiological process becomes diverted in some manner, and we have deposits occurring within the pulp tissue itself which we have fully explained in Chapter IV. If this irritation be very sudden or violent, then we have active hyperemia rapidly becoming passive, and stagnation, destroying the whole pulp, either by infarction or through the inflammatory process. It is passive hyperemia we have to fear in the

dental pulp. In a previous chapter I called attention to the fact that the pulp-chamber was normally filled with pulp tissue.

Painful Process.

In extensive hyperemia we have a painful condition, due to the swelling of the soft tissue. We have the normal process going on that we have in other tissue, i. e., the enlargement of the vessels, swelling out of the tissue; the tissue being unable to expand beyond the confines of the pulp chamber, of course make a very painful condition, due to the pressure on all the tissue confined in this canal, nerve fibers and all. A swollen or enlarged vessel must of necessity press upon those lying next to it, and thus we have pain, from increasing pressure. We find the blood vessels become much enlarged under more severe irritation (Fig. 18),

Fig. 18.
Dilated blood vessels from a hyperemic pulp. (Black.)

and by and by their contents begin to escape into the tissue through the vessel wall; not at first through a break in the wall, but between the cellular structure of it. First we have the liquor sanguinis and then the red corpuscles, by their amœboid movement, as seen in Fig. 20. Blood may pour out in considerable quantity and form a clot in the tissue and yet recover. In these cases the clot is carried away partly by the process of absorption and partly by the leucocytes, which are the scavengers of the body, for remember we stated that the pulp tissue has no lymphatics. If, however, this escape of blood be too great or for any reason recovery does not ensue, the case is liable to go through complete stagnation and a whole mass of pulp die by strangulation or infarction at the apical foramen. You will remember as we approach the foramen the root canals become constricted until it is very small at the apical opening, and it is in this part of the tooth that we have infarction occurring, shutting off completely the circulation in the pulp itself.

Discolorations.

When the plup dies from such a cause and the mass of pulp remains in the chamber a solution of the coloring matter of the blood sometimes occurs and penetrates the dentin, spreads to the enamel and redness can sometimes be seen at the neck of the tooth just above the enamel, and the whole color of the tooth is changed darker, not that the red corpuscles penetrate the dental tubuli, but rather, as I have said, a solution of the coloring matter which readily enters them. We shall refer to this fact again when we come to talk about the causes of discolored teeth. I wish to say here that when pulps in this condition die they should be removed immediately to prevent such discoloration. Thus we have seen that irritation of the dental fibrils produces hypersensitiveness of the pulp. Heat and cold cause increasing pain and add further injury, until finally the elastic walls of the vessel lose their tone, become greatly enlarged and more and more blood escapes through the walls; stagnation occurs and we have the beginning of inflammation. It must be borne in mind that these cases often get well under favorable treatment.

Symptomology.

What are the symptoms of hyperemia of the pulp, and how may we learn to recognize it?

First, get a complete history of the case; find out what caused the pain, something about the nature of the pain. In hyperemic conditions the pain is paroxysmal, usually induced by hot or cold; pain lasting a few moments, and then all is quiet until again irritated. Patient notices pain on taking hot or cold drinks, or even cold air. Unless the condition be relieved the pain soon begins to last longer each time; now an hour, perhaps next time two hours, and by and by the slightest thermal change produces the most violent paroxysms. The condition grows until death or violent inflammation takes place. Then, I say, the first thing to do is to get the history of the case; find out the things that caused pain; how long the pain lasts when it comes, and all that. Second, pain may be slight during the day when all faculties are active, using the blood supply for nutrition of the body everywhere; but at night, when active faculties are resting and the patient is in a recumbent position, pain ensues, due to increased circulation in the injured vessel, so that it not infrequently occurs that you will have patients complaining that as soon as they go to bed their tooth begins to ache; it may not ache at all in the day time. Third, in going from a cold room, where the patient has been for some time, into a warm room, pain increases; or in going from a warm room into a cold room sometimes pain increases. These are the symptoms.

Treatment.

What shall we do to relieve them? First, when we can locate the cause, remove it and put the part at rest. That is the fundamental principle in treating hyperemia. The most common cause, as I have said, is caries. If dental caries is the cause of hyperemia of the pulp, then we must remove the cause, all of it. We must remove all the decay; get it all out. I need to emphasize that fact because so many neglect that thing. They will excavate a cavity until they think it is approaching the pulp, and they leave a lot of dead, rotten material over the pulp itself and expect that pulp to get well. If the micro-organisms die and decompose there they will poison the pulp effectually, because if you have decay extending into the pulp you have an exposure in the sense of being exposed to the influences of the saliva and other substances present. Do not leave decalcified dentin that is filled with micro-organic life over the pulp for the purpose of avoiding exposing the pulp, because if the removal of it would expose the pulp then you have a pulp exposed already. When we have an actual exposure of the pulp in this way we not only have hyperemia, but we nearly always have inflammation. Provided no actual exposure occurs, you may treat the cavity with a bland, soothing agent, such as oil of cloves and iodoform.

Bear in mind that no agent should be used that will in any way irritate or poison the tissue.

After you have this done, make a tight filling of gutta-percha or some good non-conducting material, avoiding pressure upon the pulp, which requires some care when the pulpal wall is thin. Then let your case rest until recovery is complete, when you can make a permanent filling. In some cases it may require the complete covering of the affected tooth with gutta-percha for a time, as previously referred to. Most cases of this kind get well. Bear in mind that I am speaking of hyperemia of the pulp, not inflammation.

In those cases where painful hyperemia is the result of traumatic injuries, overheating by instruments, stones, burs, disks, strips, etc., resulting in thermal sensitiveness, the important thing to do is to put the part to rest. Avoid the use of hot or cold or other irritating things in the mouth; covering the tooth with a non-conductor will often be helpful; in every way protect the tooth against anything that causes pain in a given case—and recovery can be looked forward to in a majority of cases —and yet some will die, and there seems to be no way of avoiding it.

CHAPTER VI.

Destructive Changes Continued.

Inflammation. Causes of Inflammation. Symptoms of Local Inflammation. Inflammation as a Reparative Process. Conheim's Theory.

Inflammation.

Inflammation may be defined to be a morbid process going on in some tissue of the body which is characterized, when on the external surface, by its heat, redness, swelling and pain. You readily see that this definition will also accurately describe hyperemia. The first step in the inflammation process is hyperemia. Let us return to the web of a frog's foot, which we used to illustrate hyperemia. When an irritant is applied we see first a slight contraction in the vessels. Second, an expansion of the vessels; increased amount of blood in the part; vessel walls become inactive; the blood is not forced on and so continues to accumulate. The third thing we see is that the serum of the blood begins to escape; this is not coagulable; coagulable elements do not yet escape. This escape is made through the vessel walls between the cells of the endothelial lining. Fourth, the peripheral stream containing the white corpuscles begins to slow until complete stasis occurs, and here is where inflammation begins. Inflammation begins with an exudation of coagulable lymph. Remember, we stated in our study of hyperemia that the liquid sanguinis which escapes out into the tissue is not coagulable; but soon these coagulable elements begin to escape out into the tissue, and there we have the beginning of inflammation. The next step in the inflammatory process is the escape of the white blood cells. They pass through for four reasons. First, the injured vessel wall is less resistant. Second, the amœboid movement. Third, the pressure from within, the force from within, accumulation of blood and so on within the vessel wall forces it out. Fourth, by its chemotactic property. The study of the chemotactic property of cells is indeed one of the most interesting. By chemotactic property I mean the attraction which one cell, under certain conditions, has for another cell. If you watch these little white blood cells as they go out into the tissue you really feel as though they had a separate intelligence capable of directing their movement. They will wander about into the injured tissue, carrying nutrition and carrying away broken down tissue. We have two kinds of chemotactic properties—what is known as the positive chemotactic, i. e., the attraction that one cell has for another, and the negative, or the repellant action which one cell has for another.

And this is all important in the process of inflammation. Bear in mind that the escape of the leucocytes is a *distinctive feature* of inflammation, not occurring in hyperemia. Then the sixth thing we will see is the escape of the leucocytes, going on and on until the whole region is filled with these cells. (See Fig. 19.) Then the next thing that occurs is a

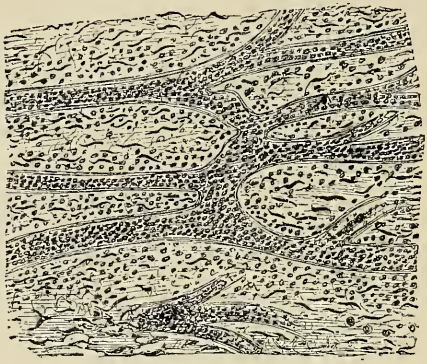

Fig. 19.

Section of tooth pulp showing the inflammatory process along the veins with diapedesis of white blood cells. (Black.)

change in the normal cell elements of the part into round cells. Nowhere can this be seen more clearly than in the tooth pulp, because nearly all of the normal cells are spindle or star shaped cells, so that the change from star shaped to round cells can be easily seen (Fig. 20).

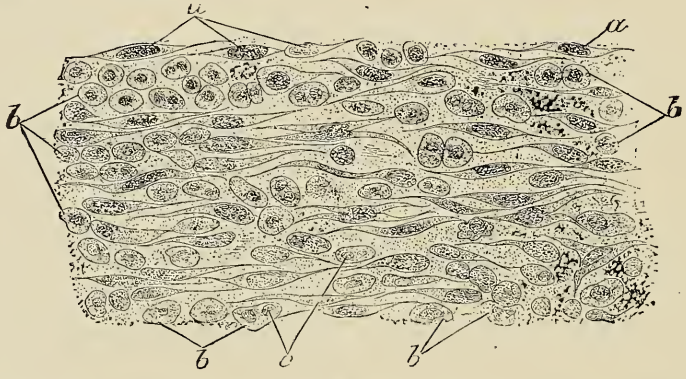

Fig. 20.

Inflammation of dental pulp. *A, a,* normal cells; *b, b, b, b,* inflammatory elements; *c,* cell dividing. (Black.)

The inflammation I have described up to this point is called simple inflammation, from which recovery may be rapid, much on the same plan as recovery is made from hyperemia. When these cells begin to change,

we have a central focus of inflammation containing coagulable lymph which creates a hard swelling. Around that we have œdema, or a soft swelling, and around that a hyperemic condition. Up to this time we have no suppuration. Then if we watch this still farther, the next element, or the eighth thing which I have indicated, is the appearance of bacteria in the region of injury, and we have a contest ensuing between the new reparative animal cells and bacteria. Sometimes the bacteria are completely digested and carried away and we have little or no suppuration, but perhaps more often these bacteria get the upper hand, the animal cells are rapidly broken down and rapid suppuration occurs. I have used this description because it more nearly represents what actually occurs in the tooth pulp undergoing the inflammatory process. For many years there was much discussion as to whether we ever had *inflammation without micro-organisms,* or whether we ever had suppuration without micro-organisms. We certainly do have inflammation without micro-organisms, as you have seen, but never suppuration without them. In the dental pulp they are most frequently the cause of inflammation.

Causes of Inflammation.

I want to say just a word about the causes of inflammation in general. We divide it into two general classes—predisposing and exciting. By a predisposing cause we mean that condition of the whole system which so acts as to reduce the resistance of the body; impoverished blood, Bright's disease, syphilis, and other forms of blood poisoning are what we mean by predisposing causes.

The second element, a perverted nerve supply. For some reason the nerve supply becomes perverted and does not perform its functions correctly.

Third, the climate.

Fourth, the age.

Fifth, lowered vitality of the cells of the part.

The exciting causes may be simple irritation, i. e., mechanical, as it were, and chemical. Cold, producing sudden changes in the blood whereby it fails to carry away its waste product, and local poisoning occurs. The whole process of taking cold is simply a process whereby the waste product of the tissue is not carried away properly, and consequently the whole system is being poisoned from reabsorption. Chemical irritations, poisons, etc., enter into this process of irritation. Then heat, as burning or scalding, as you all know.

Symptoms of Local Inflammation.

Redness.—"This symptom is persistent, and is due to hyperemia. By digital pressure the capillaries can be emptied, but on removing the pres-

sure the redness immediately returns. The shade of color depends upon the freedom from obstruction in the vessels, and the rapidity of the circulation. When the color is dark or purplish it denotes stasis; rose-red streaks along the tract of the lymph-vessels indicate lymphangitis; a dark red tract along the course of the veins would point to phlebitis; while a copper red color would denote syphilitic inflammation.

Swelling.—This symptom is due to the engorgement of the blood vessels of the part, to exudation from the blood-vessels and to proliferation of cells. In acute inflammation the swelling is soft; in the chronic forms it is hard. Swelling is especially marked in loose connective tissue.

Heat.—This symptom is most marked at the center or focus of the inflamed area. It is thought to be produced by the increased rapidity of the circulation, and the volume of blood in the part. Hunter taught that the heat of the part was never above the heat of the internal organs. Hunter's Law reads as follows: "In inflammation the heat of the part is increased above the normal temperature of the part, but not beyond the temperature of the internal organs."

Pain.—This symptom is persistent, and is increased by pressure, by motion of the part or by general exercise. Exercise increases arterial tension, and thus augments the pain. The pain is most intense in dense structures, and is mainly due to mechanical pressure upon the nerve-filaments, and is sometimes reflected to regions remote from the seat of the inflammation. Examples are, knee pain in hip-joint disease, shoulder pain in hepatitis, otalgia in pulpitis.

Disturbances of Function.—This symptom is marked in its action upon the secretions, which often become perverted or suppressed. The reflexes are generally exaggerated. Examples are the tenesmus of dysentery, the strangury of cystitis, the convulsions of teething. Non-sensitive parts become hypersensitive, examples being the pain of pleurisy, peritonitis, teething, or decayed dentine in vital teeth."—*Marshall.*

Inflammation as a Reparative Process.

I stated that following the escape of the coagulable lymph and leucocytes out into the injured tissue we see certain changes in the normal cell elements of the tissue; the normal, or star shaped cells are converted into round cells and rapidly increase in number until the whole region is filled. This increase is brought about in an interesting manner. The first plausible theory advanced, which, by the way, was the theory adopted by Virchow and Bilroth, explained the increase in the number of cells to a proliferation of the cells in the tissue. The inflammatory irritant causes the cells to take on an increased activity, attracting to themselves nutri-

ment in unsual quantity, and consequently growth and rapid multiplication (see Fig. 20 at C).

Conheim's Theory.

Conheim set aside this theory and advanced the idea that leucocytes furnished all material in the reparative process. This theory was generally accepted for at least two decades. He attempted to prove that the cells of the part did not undergo any active change during the inflammatory process except degeneration or breaking down. Conheim claimed that the normal cells of the tissue took no part whatever in the reparative process; the only change that took place during the inflammatory process in these cells was that of degeneration. The manner in which he attempted to prove this was indeed very interesting. For this purpose he took the cornea, because of its transparency. When he treated the cornea with chloride of gold a most perfect network of stellate cells appeared lying in the inter-cellular substance, which suggested to him that these spaces, i. e., these inter-cellular honey-combed spaces, furnished opportunity for the migration of these wandering leucocytes, i. e., they were able to migrate out through this inter-cellular substance. Seeing these apparent wandering cells lodged therein he determined that it was a process of passing through the tissue which he saw, and consequently he believed that they were the agents which had most to do with the reparative process. The following was his experiment: He drew a ligature through the bulb of the eye of a rabbit. In 24 hours the transparent cornea became opaque, due to the increased number of leucocytes which were present, as shown by taking the cornea before it became opaque and examining it under a high power. He claimed that all the new cells were identical with leucocytes, i. e., the new cells that he saw in this cornea. To further prove his point he injected small granules of carmine, held in suspension, into the lymph sacs and the blood vessels of a frog; he then produced his inflammation of the cornea and many of the new cells which he found contained these granules, which, by the way, he was unable to find anywhere else in the system. These experiments were performed in 1867, and they created the most widespread discussion everywhere. Scientists everywhere were discussing this theory of Conheim's, and as a result it created a great amount of opposition. Many men attempted to prove the old theory of cell proliferation, and *Strycher,* of Vienna, took up this theory and advanced it still further, and evolved his theory of tissue metamorphosis in which he claimed that not only did the cells, but the entire tissue, inter-cellular substance, etc., all return to their embryonic condition. He took up this theory of cell proliferation and did an immense amount of work. Few of us can com-

prehend the amount of work these scientists did along these lines, trying to prove that not only the cell elements, but the inter-cellular substance and all returned to its embryonic condition, from which it rapidly grew, and separated into amœboid masses from which new tissue was gradually developed. From that time on men everywhere have been studying this process.

Grawitz's theory was based on the fact that throughout all tissue in the inter-cellular spaces are seen slumbering cells, i. e., the cells which Conheim claimed to be leucocytes, but which Grawitz claimed were really slumbering cells, which under certain conditions are capable of waking up, as it were, and undergoing certain changes. By their amœboid movement they wander out through the tissue, taking on active life. At first the slumbering cells have but a small nucleus and little or no protoplasm. Their nuclei gradually enlarge and acquire a cell body and assume all the functions of tissue cells, with protoplasm and all. This, then, was the cell, according to Grawitz, which produced the process of repair. At the present time, scientists account for the presence of such a large number of cells in reparative inflammations to all three of these sources. The normal tissue cells multiply, in three ways.

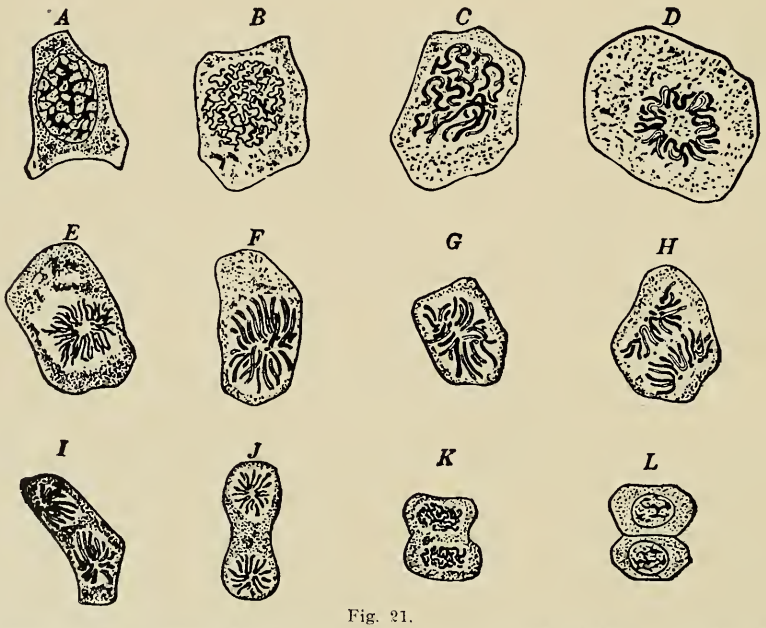

Fig. 21.

Karyokinesis. A, resting stage; B, convolution stage; C, wreath stage; D, monoster stage; E, lengthening of cells toward the poles; F and G, the rosette dividing into two groups; H, the two groups pass to opposite poles; I, diaster stage; J, diaster stage, with complete neuclei; K, convolution stage, daughter cells; L, daughter cells at rest.

First, by budding.

Second, by direct cell division (see Fig. 20, c).

Third, which, of course, is by far the most interesting and the one that is the most common, most usually known as karyokinesis, which I have attempted to illustrate in Fig. 21. That is the process by which the cell elements of tissue reproduce themselves mostly.

The second element in this inflammatory process of repair, the slumbering cells awaken into new activity. These cells resemble the leucocytes in appearance, and it is doubtful if one is not taken for the other. These cells lie caught, as it were, in the network composing the normal tissue. Many consider them purely embryonic cells left from the embryonic condition of the tissue. In the growth and development tissue maintains largely an embryonic condition until maturity. During all of this developmental stage a greater amount of these cellular elements are required than after maturity is reached, consequently, the theory is that this excess of cells that have not been used atrophy and are caught and lie there in the inter-cellular spaces—one of the wonderful provisions of Nature; they are a reserve fund, kept there to be used in time of need. They are used in time of over-exertion of muscular tissue and during inflammatory processes always. That is the second element as seen in tissue repair.

I stated that when they are first seen they scarcely seem to have even a nucleus—simply a small speck, with no protoplasm. When they are brought into action they gradually enlarge their nucleus and take on forms of protoplasm. Protoplasm furnishes the means of carrying, shall I say food, to build up the waste tissue.

Then the third element that we account for in this process of repair from inflammation—the leucocytes normally floating in the blood stream are attracted there for reparative purposes. They bring fresh nutriment and more especially carry away useless, broken down material, waste tissue, etc.; that is the greatest function of these leucocytes which normally float in the blood stream. What other function they perform we at present haven't a clear idea. We thought we had a few years ago, but that whole notion has been changed now. There is an actual increase of white blood cells during inflammation, not only out in the injured tissue, but also in the whole blood stream. The whole system thus appears in sympathy with the injured tissue and those organs in which the white blood cells normally abound and perhaps have most to do with their formation—especially the spleen and lymphatic glands—are much enlarged during the process of severe inflammation, due to reflex action from the injured arterioles. The greater the injury, the larger the number of cells. Slight inflammations require but a few in the process of

repair and Nature provides but few. Severe inflammation and suppurations requiring more, Nature provides more. Those white blood cells which have to do with the carrying of food material, so to speak, and the carrying away of waste material, we call phagocytes. The most interesting process taking place in the animal body is that of metabolism. All higher forms of life are but many simple cells so arranged as to form a structural unit. These cells are only capable of growing under favorable conditions. The whole mechanism of the body is a unit of cell collections; in order for these cells to grow and perform their function, they must have food; they must grow and reproduce themselves. You can readily see that if these cells did not constantly reproduce themselves the whole body would soon die out.

Third, they form waste products which must be secreted. When food is taken into the stomach, having previously been acted upon by the saliva, it is further acted upon by the gastric juices, and part of it, which is now ready for absorption, passes directly into the blood through the walls of the stomach. The remainder passes on to the intestines, and is there again acted upon and absorbed directly into the lacteals; from there into the blood. This blood is then taken to the lungs and to the liver, respectively, and then passes directly to the tissue. Each time it enters the blood it unites into chemical combination with the protoplasm of the cells.

The process by which all of this is carried on is a chemical process pure and simple, and when the blood passes through the lungs it adds oxygen, as you know, and we have a highly complex substance of many chemical elements united in such a way as to be readily exchanged into cell tissue. At the present time the chemistry of the proteid molecule is poorly understood. There is room for some of you gentlemen to do some splendid work along that line, and it is the only thing we need now to perfect us in many lines of medication. Just as soon as we are able to know definitely the chemical combination of this proteid molecule, just so soon will we be able to know what substance we can administer that will chemically unite with it to change it in the direction we wish it changed. The process, then, by which this nutrition is taken up in the cell—the phagocytes bring this material in its most absorptive form, the form in which it can be most readily used by the cells of the tissue. Then by the process which I spoke of as the chemotactic property, cells in certain conditions are attracted to cells in certain condition, chemically. The phagocytes containing certain chemical elements are attracted to the tissue cells containing certain chemical elements. These chemical elements are constantly changing in the tissue cell, as well as in the phagocytes. The process of wear on tissue cells is a chemical process. Fatigue is a

chemical process in the cells themselves. We are just beginning to realize this. It is the all important phase of physiological chemistry.

The waste products that these cells form—speaking now of the tissue cells—has to be carried away. The waste product is largely urea and uric acid, and one of the functions of this phagocyte is to exchange its nutriment for the waste product of the tissue cells. It is carried away by way of the kidneys and excreted in the form of urea, and excreted partly by way of the lungs in the form of carbonic acid. That is the whole chemical philosophy of the system, and nutrition is purely a chemical process. I hope I can firmly impress that upon your minds.

I think I have said sufficient to enable you to follow my further illustrations. Take a simple incised wound. The edges are covered over with this coagulable exudate and clot, rich in albumen. I might say that in olden times the surgeon was governed in his treatment of these wounds by the presence of this coagulable exudate upon the surface of his incised wound. He waited until that appeared, as he thought, good and healthy, then he brought his wound together, believing thereby that he would cement the wound by bringing these two coagulable exudates together. Of course, the method which we now follow is to bring our incised wound in direct opposition. If we can do that we have our slight exudate forming, filling in the mechanical defect. Then these cells abounding in this exudate, being transformed into embryonic tissue gradually change into permanent tissue form, into which we have the little vessels sprouting out, as it were following out into the tissue itself, until finally the blood vessels unite on opposite sides and we have the circulation re-established, and lastly we have the epithelium growing out and covering over the wound. Take a wound where a large surface is involved, where it is not possible to bring all the surfaces of the wound together; the healing process is brought about by the building up of this embryonic tissue from the bottom until the whole tissue is filled up; then we have the epithelium reproducing itself, quite in the same manner that ice freezes over a pond, first beginning at the circumference, then a little farther in and so on until finally it covers over the whole mass. All of these cells, of course, as the change is made from embryonic to permanent tissue, assume the spindle shaped form and become fully organized into fibrous tissue. The first process that I outlined to you is what is known as healing by first intention.

Chronic inflammation is a condition in which we have the symptoms of acute inflammation, but to a less degree. It usually follows an acute attack, and may continue for years without much apparent change. Usually the tissue hypertrophies—we have fungous growths, elephantiasis, tumors, etc., but more frequently is the end of acute inflammation and passes directly into suppuration.

Treatment of Inflammation.

General Considerations.

The first principles in the treatment of any inflammation is to remove the cause and to put the part to rest. If the cause be systemic, then we must have that corrected first; if due to irritation of any kind that must be removed. This irritation may be of such a nature as to require systemic medication; indeed, in all severe inflammations the eliminative organs should all be stimulated. The bowels and kidneys should receive special attention as well as the skin. In addition to administering cathartics, diuretics and diaphoretics, the frequent sponging the surface of the body with tepid water and drinking large quantities of water will prove most valuable. The application of ice, cold water, either sprays in the early stages of inflammation before stasis has occurred, especially where heat and redness is pronounced, is considered most potent for good.

Heat in the form of compresses wrung out of hot antiseptic solutions, changing as often as they become cool, is a very excellent method of stimulating the collateral circulation and relieving the pain. When the pain is severe it may be necessary to resort to the use of some of the coal tar derivative anodynes, narcotics or opiates. What is meant by putting the part to rest is best illustrated in the prevention of occlusion of a tooth that has become sore from pericementitis or excluding the light from an inflamed eye. Among the other local measures that are of value blood letting, scarifying, cupping, the use of leeches and the application of counter irritants, are suggested, especially over deep seated inflammations. When the blood pressure is high and the pulse very rapid the use of circulatory depressents are indicated.

Symptoms of Inflammation of the Tooth Pulp.

There are no special symptoms of inflammation of the tooth pulp; they are those of hyperemia in a more aggravated form, the paroxysms of pain last longer and are more intense. We have some difficulty in making a differential diagnosis of inflammation because we cannot see the swelling, the redness, etc., that we can see on external surfaces in the soft tissue. Pulps often become inflamed, suppurate and die without any pain whatever. In hyperemia there is no tissue change. All the cells in the tissue are unchanged, except the walls of the vessel; while in inflammation all the changes are taking place as I have described, namely, the presence of coagulable lymph, the white cells out in the tissue, etc. Up to this point recovery is possible, and, indeed, this stage is about the healing process of all simple inflammations, such as the healing of surgical wounds. Remember, simple inflammation is always a repara-

tive process. All tissue injury is healed by this process, which is purely physiological. Inflammation of the tooth pulp occurs, after that organ is exposed, either from decay or other cause. More frequently it is caused by caries that has penetrated to the pulp, or at least the poisons of micro-organisms in the carious cavity penetrating to the pulp. We doubtless do have inflammation of the pulp without micro-organisms being present in the tissue, as we have simple inflammation anywhere. It is not always possible to tell just where we have hyperemia conditions and where we have inflammation. We can only decide as we work at the cavity, removing the carious dentin. We may have only a slight portion undergoing the inflammation process immediately around the point of exposure, and the rest of the pulp remain practically healthy, perhaps a little hyperemic immediately around the point of inflammation. But as time goes on and exposure to saliva and micro-organisms continues, we will see the inflammation spreading in the direction in which the poison is carried by the circulation.

By and by suppuration occurs; not in all cases. Some exposed equally long do not suppurate; due, doubtless, to the condition of the cells of that organ, and perhaps somewhat to the nature of the irritant. This will be referred to under the head of suppuration. After securing the history of the case we must depend upon what we find when opening the cavity, to determine our condition. Does decay penetrate the pulp chamber? If so, we have a real exposure. What is the condition of the decay? Is it black, hard, dead decay in which the former life has died and passed away largely; has it progressed slowly; does slight pressure on the pulpal wall cause pain; has the pulpal wall been broken through; is there growth of the pulp tissue out into the cavity? These are the things that aid us in determining upon the correct diagnosis.

Hypertrophy of the Pulp.

In inflammatory cases where suppuration does not ensue the pulp oftentimes seems to grow rather than break down. There is a continual growth of new cells, just as in reparative inflammation in healthy tissue. These cells of repair continue to divide, after forming new nuclei within themselves, until we actually have a growth of new tissue out into the cavity of decay (see Fig. 22).

In a cavity of this kind the pulp is actually pushed into it. Oftentimes the odontoblasts and fibrils are pushed out also. In other cases the growth is within the tissue itself, and not simply the exposed point. These cases are not usually painful, for the reason that there is a way of escape for the swollen enlarged tissue.

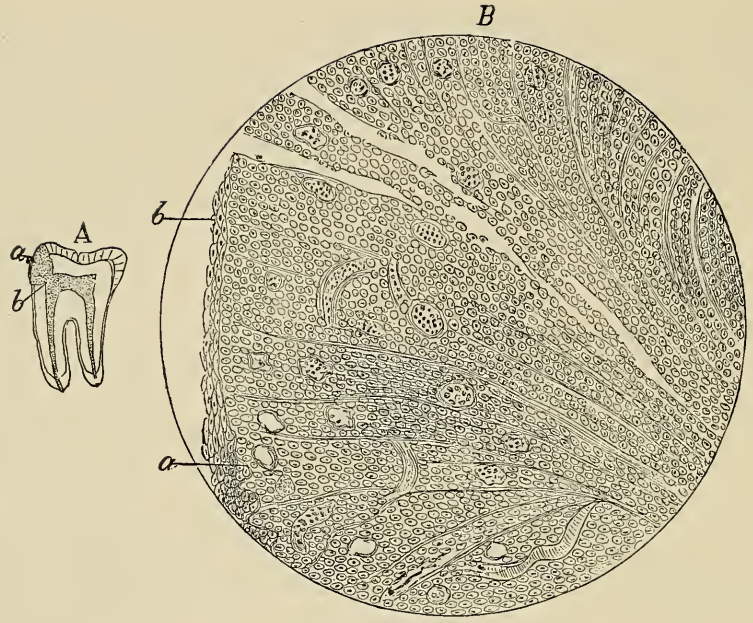

Fig. 22.

A, diagram of lower molar with cavity at *a* completely filled with hypertrophied pulp tissue grown out through opening into pulp chamber at *b*; *B*, showing granulation tissue of hypertrophied portion; *a*, epithelial cells with papilla; *b*, epithelial cells without papilla. (Black.)

How to Study Hyperemia and Inflammation In Tooth Pulp.

A word as to how we study the conditions of hyperemia and inflammation in the dental pulp. I will give you a method which has been quite universally adopted of late, a method suggested by Dr. Black. That is first to get the patients while they are suffering from the paroxysms of pain. If the paroxysm has passed over, wait for another attack, or excite it by thermal changes; then while the pain is at its height extract the tooth and immediately drop it into Miller's fixing fluid.

The object is to capture the condition, as Dr. Black states, and harden the tissues so it can be handled without in any way disturbing the contents of the vessels. Then it is broken open in a vise and the whole thing dropped into Miller's fluid again, and while under this fluid the pulp is separated out and lifted out of its bed. Oftentimes you will lift the pulp with the odontoblasts and the fibrils attached to it, some of them. Now leave in Miller's fluid for farther hardening, after which it is dropped into a solution of gum arabic which has been thickened by slow evaporation. Of course the object of this is to fix it so it will be hard enough to handle. After 24 hours it is put in wax, and after 12 hours it is cut in

the ordinary manner of cutting specimens, mounted and examined under the microscope, with the results that I have given here.

Causes.

There is very little that can be said as to the causes of inflammation in the tooth pulp other than what has been said regarding hyperemia, to which the reader is referred. Indeed, as has been said, inflammation usually begins in hyperemia, and the reader must bear in mind the fact that all severe hyperemias are liable to run into inflammation, and unless steps are taken early to prevent this, the vitality of the pulp will have to be sacrificed. Bear in mind that inflammation rapidly follows pulp exposure from caries or accidents in excavation; but inflammation of the pulp does not depend on these, but the same variety of causes that have to do with hyperemia obtained as well.

The use of corrosive agents, shock, lowered vitality of the general system have an important bearing on inflammation in the tooth pulp, the same as in other parts of the system.

Treatment of Inflammation of the Tooth Pulp.

Regarding the treatment of pulp inflammation the same principles that are carried out regarding other inflammations elsewhere about the body must be followed here, which relates to removing the cause and in putting the proper part to rest. We must remember that intense pain is usually present, and it is our business to relieve this as quickly as possible.

If irritation through a carious cavity is the cause, then that should receive our first attention. The method I follow in this regard is to carefully break down the overhanging enamel and wash the cavity thoroughly with warm antiseptic solution, after which I apply the rubber dam and dry the cavity, using dehydrating agents and warm or cool air, whichever feels more comfortable to the patient. I next excavate the carious dentine, proceeding in such a manner as to produce the least possible pain; this usually can be accomplished best by removing the decay from all walls around the pulp first, leaving the pulpal wall to be removed with one stroke of the excavator. In the use of our excavators if we cut from the pulp rather than towards it, we are less liable to injure it and to produce pain.

In making the excavation if the pulp is exposed so as to bleed the pain will be relieved thereby in most cases. If exposure does not occur in this manner it is regarded as good practice to open the pulp, provided this can be accomplished without causing too much suffering. Oftentimes this can be accomplished with the aid of warm carbolic acid, cocain or other local anesthetics, and some operators administer nitrous oxide.

and remove the pulp immediately. I wish to emphasize the value of blood letting whenever severe pain is present. The next step is to apply some mild antiseptic soothing agent and seal the cavity in such a way as not to produce pressure on the pulp and at the same time preclude any possibility of the patient forcing the stopping down against the pulp in the act of masticating. The hot foot bath will serve to attract the force of the circulation from the head and thus relieve the pain. A little mustard added to the hot water will increase its efficacy. The administration of saline cathartic is commended in severe cases, especially where there is some tendency to pus formation. For the purpose of relieving pain it is occasionally necessary to resort to the use of some of the narcotics and heart depressants. It is not considered good practice to attempt pulp devitalization in these painful cases until the pain has been relieved and the patient made comfortable for at least forty-eight hours. This fact will be alluded to under pulp devitalization.

CHAPTER VII.

Pulp Capping.

History. Favorable and Unfavorable Cases. Methods of Pulp Capping.

History.

In looking over the literature I find that men have long been capping pulps. I find records of pulp capping as far back as 1850. In that early day men knew nothing scarcely of how to treat diseased pulps and fill root-canals, so palliative measures were used until the aching tooth was quiet; then fillings were made, sometimes without even lining the cavity. Most of their cases died and suppurated. Then they either bored a hole underneath the gum margin or extracted the tooth. A little later than this, in about 1858, men began to cap pulps with oxychloride of zinc, and from that early time until this the subject has engaged the attention of many experimenters. Almost all sorts of materials have been used—lead, tin, asbestos, varnishes, gutta-percha, oxyphosphate, all of these have had their adherents. In 1888 I remember hearing this subject discussed before the American and Southern Dental Societies, which met in joint session in Louisville. There seemed to be a very unanimous opinion then that failures were had more frequently than success. I remember well the remarks of Dr. Storey, of Dallas, Tex. He stated that he had taken up the then prevalent fad of capping pulps and had used all the accepted materials, and in the next few years had more business than he could possibly attend to, and most of it was caring for putrescent pulps and abscesses in teeth whose pulps he had previously successfully capped. While this has not been my experience, yet I have capped many pulps, a majority of which have been failures. I have tried to study out the causes of these failures, with the result that I now cap comparatively few exposed pulps, and those under the most favorable circumstances, which circumstances I have already stated to you. Many men report successful cases of capping who have not the opportunity of following them up. Cases leave us when we are unsuccessful and we never learn of our failures. I know I am called upon frequently to treat cases where pulps were previously capped by others. These cases sometimes go on for years and give no trouble. I have had cases under direct observation for three and four years; I was able to know that the pulp was alive during that time, and then all at once, without warning, trouble begins.

There are men who claim to be able to remove a portion of the pulp

surgically, as it were, and cap the remaining portion. They sometimes remove the bulbous portion in the pulp-chamber and the contents of one of the root canals in a molar and cap the remainder. You will find as you read the literature on the subject that men have actually practiced this sort of thing, not only practiced it, but advocated it for years. I must say that it will require much stronger evidence than I have seen to convince me that the remaining portion lives in a healthy state any length of time.

Favorable and Unfavorable Cases.

When to cap a pulp and when to destroy it are questions which can only be decided after considering a great many things, among which are the following:

First—The exposure. Is there an actual exposure? Has the carious process exposed the pulp? Is there only a slight exposure in the horn of the pulp? Was it exposed in removing the decalcified dentine? Was it exposed by an accidental slip of the instrument? These conditions can all readily be observed after the tooth has been cleaned, washed and dried. If there is any doubt, slight pressure on the pulpal wall with a small ball of cotton or round burnisher, when, if exposed, pain will be felt by the patient.

Second—Has the pulp been infected? This is usually the case if caries has penetrated directly to it, or if exposed by accident, which sometimes unavoidably occurs. Infection is very likely to result if exposed to the air for any length of time.

Third—What is the history of the case? Has the pulp caused pain to any extent? Has it been hyperemic or congested at any time? These are all unfavorable indications, and failure will surely result from attempting to cap such pulp, no matter what method is followed.

Fourth—Has the pulp been actually injured? How?

Fifth—What is the history of previous cappings in the same mouth? Had the patient the unpleasant experience of having pulps capped under similar conditions and then die with all the pain of an acute alveolar abscess resulting therefrom?

Sixth—Has the tooth fully developed? It is so important to preserve the vitality of the pulp until the tooth is thoroughly formed that I sometimes take a chance even when certain conditions are inclined to be unfavorable, for to destroy a pulp in a partially formed root means the loss of that tooth sooner or later.

Seventh—What is the general health of the patient? I have poor success in capping pulps for anemic individuals, those suffering from poor elimination, or those of nervous, hysterical makeup.

Eighth—Is the tooth situated in the anterior part of the mouth, where the natural translucency of the tooth is very desirable?

Ninth—Is is not desirable to cap pulps that require much medication to restore them to comfort?

These are some of the questions which must pass through your mind before you should decide either for or against capping. There is still another important point to consider in this matter, and that is with reference to the personality of the patient. Is she one of those who will unreasonably blame you if failure results? There are a few people who feel that when they have a cavity filled that should for all time end their trouble with that tooth, and if the pulp should die in such a tooth they would condemn the dentist both loud and long as an impostor.

Methods of Pulp Capping.

In preparing pulps for capping it is desirable to free the cavity from all poisonous material and cut to sound dentine upon which to rest the periphery of the capping. If this does not actually uncover the pulp so much the better, but in any event it is important that there should be no space between the cap and the pulp. It should lie down on the pulpal wall, or in case of an actual exposure down on the pulp itself with no space for air, secretions or excretions from the pulp, and yet this must be done without the slightest pressure upon that organ.

Materials.—A great variety of substances have been suggested as pulp cappings, many of which have been tried and discarded. Oxychloride of zinc mixed into a thin paste, dropped on one of the walls of the cavity and coaxed over the pulp in such a manner as to exclude the air has been used by some for many years. Others cut a piece of white linen paper just large enough to cover the pulpal wall and put the zinc cement on this and carry one end to place, then gently press from this towards the opposite end, forcing some cement ahead of your pressure and out around the periphery. Many advocate zinc oxyphosphate cement used instead of the oxychloride, and others the oxysulphate of zinc. Zinc oxide cement powder mixed with oil of cloves or other similar oil has many advocates. Some practitioners use iodoform 10 per cent with the cement powder, then mix either with oil of cloves or the cement liquid; in all of these the method of capping is the same. Instead of the white linen matrix many use gutta-percha disks cut small enough and depressed slightly in the center of the surface to be placed next to the pulp; on this depressed surface is placed some one of the above mixtures and carried to place, as before described. Small metallic concave disks have been made for this purpose, and have the advantage of being more convenient of application. Solution of gutta-percha in chloroform and

some of the balsam varnishes in alcohol, to which may be added oil of cloves, iodoform or some other agent, have been suggested. These solutions are used on the little disks in the same manner as the cement mixtures. A word of caution needs to be given regarding the use of zinc oxide; many specimens have arsenic present in them which should be avoided.

Insist on having a pure oxide. Dr. A. E. Royce has recently suggested incorporating 5 per cent hydronaphthol in the cement powder, and from this make a mix, using the same means of applying that I have already suggested. I have had very satisfactory results from this mixture, that is, if an opinion formed after two years of frequent using is of value.

It is the usual practice to complete the filling in all these cases either with gutta-percha or oxyphosphate cement—especially a layer of cement over the capping. If the tooth remains normal for a period of six months it is usually considered proper to place the permanent filling.

I think it wise to add this further word—all modern pathologists regard vital pulps that are normal to be of great advantage to teeth as regards their resitance to decay, their color and general comfort to the patient.

In concluding this subject I wish to say that when you have used your best judgment both as to the case, the materials, and the method of doing the operation, sometimes failures will result. Some cases will go for years without the least discomfort to the patient, and all at once take on violent inflammation; others die and give no trouble, and still others start up trouble immediately after the capping is placed, which increases until death of the organ results either of itself or at the hands of the operator—so that we must not be too sure of success, and yet this operation is done successfully often, and under favorable conditions in a great majority of cases.

CHAPTER VIII.

Pulp Devitalization.

Methods. Preparation of Cavity to Receive Arsenic.

Devitalization of pulps is a subject of increasing interest. Many teeth come under our care that have passed beyond our ability to save with pulp alive, and have them remain so for any length of time. We devitalize pulps for the following reasons:

First. Inflamed, aching pulps that have gone beyond conditions favorable for capping.

Second. Cavity may be so shaped or caries progressed so far as to make permanent filling impossible without anchoring in the pulp-chamber.

Third. A crown may be necessary, either to restore a broken down tooth or as an abutment for a bridge. In these cases it is seldom possible to properly prepare for a crown without removing the pulp, on account of its sensitiveness and the danger of approaching too near the horn of the pulp; also danger of exposing the entire dentin and fibrils to severe irritation of large amount of zinc cement, thermal changes, etc., etc. I am not one of those who claim that it is impossible to occasionally fit a crown for an abutment of a bridge without devitalizing the tooth; I believe there are many teeth where it is possible to do that, but in the great majority of cases it is not possible. It is claimed by some that when teeth are ground in this manner for the adjustment of crowns and bridges, and the pulps left alive, that the action of the arsenic contained in the cement will eventually destroy the pulp. I have never been able to get interested in the theory of pulps dying under the arsenic contained in the cement. I never felt that that was sufficiently proven to be taken as a fact.

Fourth. We devitalize pulps in teeth in advanced stages of pyorrhea alveolaris.

Fifth. We devitalize pulps where the patient is suffering from calcific deposits within the pulp itself. Oftentimes, as I stated before, this occurs in teeth that are perfectly sound, so far as we can tell, and our only method of getting permanent relief for the patient is to devitalize and remove the pulp.

Before attempting to devitalize we should first restore the tissue to a normal condition, so far as possible. Inflamed, aching pulps need some palliative treatment first, either by actually exposing the pulp and letting out some of the blood, or in case the pulp is very hypertrophied, with

the use of a little carbolic acid and cocain we cut off the hypertrophied portion before attempting to apply the devitalizing paste. For the palliative treatment we usually use oil of cloves, carbolic acid, creosote, chloroform and some of the oils, cocain and warm oil of cloves, morphia, laudanum, alcohol and the essential oils; any of these sealed in without pressure, and left for a day or two until the pain has subsided. The reasons for doing this are two. First, an inflamed pulp is very resistant to the absorption of devitalizing agents. That is an experience that I am sure all have had who have attempted to devitalize aching pulps. Second, our devitalizing agent acts as a further irritant and sometimes causes intense suffering needlessly. In cases where there is only slight sensitiveness and the pulp has begun to suppurate in its horn, I have had best results by letting out the pus, washing out freely with warm antiseptic solutions, using then a good antiseptic or germicide to do away with the suppuration, and then proceed to devitalize and remove the living portion.

Care must be taken not to mistake pressure on the pulp-chamber content for irritation to the fibrils. That is a mistake that is made very frequently. When we come to excavating close to the pulp we will find that we give pain oftentimes when really the pulp itself is practically dead. The reason for this pain is that the chamber is filled, literally filled full of material, and the slightest pressure upon it produces irritation beyond the apex. You will often open up teeth, even after you have applied your devitalizing paste, that seem sensitive upon excavation, when with a little care you can succeed in exposing a little corner of the pulp, and after you have done that you can proceed to open it completely and remove it without any pain whatever.

The object in devitalizing pulps is, of course, that they may be removed painlessly. I might say that dentists oftentimes really forget the object of devitalizing pulps, and proceed to half devitalize and remove them with as much pain as if they hadn't attempted to devitalize at all.

Methods.

Orangewood stick. The oldest method so far as I can learn that is practiced to any extent at the present time is called knocking the pulp out. For this method the pulp-chamber must be thoroughly opened, the entire pulpal wall removed, a piece of orange or rose wood is whittled quite like a sharpened lead pencil approximating the size and shape of the root canal, this is dipped in carbolic acid, and held in direct line with the pulp the point touching it, the stick is then struck a quick blow with the mallet. If everything works well, the pulp can be removed quickly and in some instances painlessly, but many unlooked for things may happen such as breaking the stick, driving it through the apex, or failure because

of the irregular shape of the canal. Then the pain caused in so thoroughly opening the chamber is frequently severe. Altogether I regard this as a relic of barbarism, which should be forgotten, and yet some good practitioners use this method occasionally. My only excuse for presenting the subject is its antiquity.

Carbolic acid method. Carbolic acid has been used for many years as a corrosive agent to destroy pulps little by little, requiring frequent applications, and many days' time; but more recently it has been used by hyperdermic injection directly into the pulp tissue using a very fine needle carried up along the wall of the chamber for nearly half the length of the canal, then forcing a drop or so of the melted crystals into the pulp. In a few moments it can be removed quite painlessly, the pain of opening the chamber and introducing the needle are often as great as to remove the pulp forcibly without it.

Cocain. Many methods of using cocain for the purpose of anesthetizing the pulp have been tried, only two of which seem to be used at the present time—viz., the cataphoric electric apparatus, and pressure method used in exactly the same manner as has already been suggested in Chapter II. for obtunding sensitive dentine, with addition of forcing the cocain so thoroughly into the pulp tissue that all sensation is lost. The objections to cocain extirpation are these:

First. The danger of forcing poison of some nature through the apex and injuring the tissue beyond.

Second. The injury to the tissues in the apical space caused by tearing the pulp away.

Third. The hemorrhage that usually follows.

Fourth. The soreness attending the absorption of the blood clot left in the apical space.

Fifth. The danger of leaving a small fragment of pulp tissue.

The advantages are these:

First. The time saved; operation can be completed in one sitting.

Second. Less liability of tooth discoloration.

Third. In many cases less painful than the arsenic method.

Cocain method. The pressure method seems to have entirely displaced the cataphoric electrolysis. The first essential in the use of the pressure method is a clean cavity, so shaped that cocain solution can be confined under pressure—this will often necessitate the building of a third wall, to make the cavity nearly cup shaped. After the pulpal wall is obtunded it should be thoroughly removed before continuing pressure to fully anesthetize the pulp in order to avoid forcing micro-organic poisons into the tissues beyond; then the pumping can be done with some such instrument as is represented in Fig. 23A, or a piece of soft rubber a little

larger than the cavity, and forcing the cocain solutions into the tubuli by pressing this against the cotton carrying the solution, with a sort of pumping motion, gently at first and then increasing gradually. This pumping should continue until all signs of sensitiveness are quite gone. This pumping should begin gently then with increasing force until considerable force is exerted. A smooth, fine broach should be passed along the chamber wall, and if slight sensitiveness is found the cocain should again be pumped—until the smooth broach will pass to the apex without causing pain which should require not more than five minutes. A barbed piano wire broach which has been selected for the case, tested and sterilized should be carried well into the canal, turned half a revolution and withdrawn, when in most cases the pulp will come away entirely. Care should be exercised not to cut or tear the pulp tissue, roughly forcing in the broach, or turning it too much and cutting the tissue into little pieces. When the pulp is torn in this manner it is almost impossible to remove all the shreds before sensitiveness returns, and to remove these shreds is a task that sometimes puzzles the most skilful; in my hands the most successful method of doing so is by entangling them in cotton loosely wound on a broach and dipped in 95 per cent carbolic acid.

Fig. 23A.
Tuller's cataphoretic instrument

For the purpose of controlling and preventing hemorrhage a number of hemostatic agents have been suggested but the use of 1-1000 solution of adrenalin chloride as a vehicle for dissolving the cocain crystals seems to be most often used, and in cases where there is no hyperemia of the tissue in the apical space, seems to meet every requirement. We should bear in mind that most cases calling for pulp removal are those that have recently been in a state of inflammation, and it is almost certain that a hyperemic condition exists beyond the apex, in which case a reasonable amount of

bleeding will be helpful and should be encouraged. So far as using blood coagulating or clotting agents is concerned for the purpose of stopping the hemorrhage after pulp is removed, I wish to ask what is to become of this clot which to be of any value must be beyond the root canal foramen? Clearly it must be absorbed or organized and is this not a source of danger? I think it best to wait a few minutes on nature, and let her stop the bleeding by closing the lumen in the broken vessels. The next step is to remove the blood mechanically, with aseptic cotton and proper broaches, and finally with alcohol. Dr. J. P. Buckley calls attention to the error of using hydrogen dioxide for this purpose; it tends to discolor the tooth substance. After thorough dehydrating some mild soothing agent should be sealed in for a few days, to allow nature to heal and restore to normal the tissues about the root apex.

For this purpose I use a mixture of eucalyptol, oil of cloves and trikresol, placing the smallest quantity possible on antiseptic cotton carried well into the canal. The reasons for not filling the canal immediately upon the pulp removal, are two; the tissues beyond are more or less anesthetized and consequently will not respond in such a way as to tell you when apex is just closed, and no filling material forced beyond and second, there is some liability of leaving a tiny shred of pulp tissue at the apex, which will not only prevent thorough filling, but will afterwards cause considerable pain when the anesthesia has passed.

If we will keep all these suggestions in mind, I have no doubt we will find the removal of pulps by this method very satisfactory in most cases, and perhaps the most satisfactory of all methods, all things considered, for all single rooted teeth in the mouths of the average patient, but in three rooted teeth, especially where some of the canals are very small, and in the mouths of very nervous people, the arsenic is preferable.

Arsenic method. The standard method, the one most frequently used, and of most general application is the arsenic method. Arsenic acts by first exciting the sensory nerves and then paralyzing them. It always arouses a degree of inflammation somewhat dependent upon the amount used so that it is advisable to use the least possible quantity to accomplish the desired result. While sensation is somewhat paralyzed a few hours after the application of arsenic, yet the tissue is not dead, or even senseless for several hours after application. Arsenic causes death by its irritant corrosive action; death by infarction in the apical portion is a result of the inflammation caused. Inflammation may be so severe as to prevent the ready absorption of the arsenic and hence death will be very slow in such cases, and usually attended with considerable pain. This emphasizes the folly of attempting this method where pulp is in an inflamed condition. If the pulp is quiet and small quantity used in con-

junction with soothing agents the desired result can be attained without pain, indeed, in my own hands it is rare indeed that I have trouble of this kind. The preparation I use is made as follows: Arsenious acid and finely powdered cocain hydrochlorate are taken in equal quantity and thoroughly rubbed together, after which sufficient oil of cloves is added to make a thin cream; to this mixture I add one-half millimeter squares of white hard blotting paper until the cream is absorbed. In a few hours these will dry sufficiently to put in a jar and not stick together.

The tiny squares can be carried with the pliers and does away with all danger of getting arsenic anywhere but at the point desired. The brown color which they soon assume is an added advantage. It is advisable to place arsenic preparation directly over an exposure of the pulp, but this is not absolutely necessary for it will cause death of the pulp when applied just beneath the enamel if long enough time is given—but the danger lies in tooth discoloration from solution of hemoglobin in the blood—and yet this danger is not so great as make exposure an absolute necessity, when that can only be accomplished at the cost of a great deal of pain. As stated before the object of devitalization is that the pulp may be removed painlessly and if we cause much pain in applying the remedy we have not accomplished the thing desired. In most cases calling for this treatment the pulp is already exposed by caries, and is only covered by decayed dentine which can easily be removed by following the method in excavating already suggested. After the preparation is placed it should be covered with some material that will make a perfect seal and yet not press on the tissue.

Preparation of Cavity to Receive Arsenic.

A very important point to consider is the proper preparation of the cavity—it must have sound walls and margins, particularly at the gingival—and so shaped that the sealing will not be driven out or down in the act of mastication.

In very many proximal cavities the gingival wall is under the gum gingivus and indeed that tissue is very often grown into the cavity, which requires special care in removing or by wedging back. In all cases the rubber dam should be applied and the field of operation made clean surgically. Then a properly fitting matrix can be placed, and a fourth wall built of cement or in favorable cases gutta-percha, never temporary stopping, may be used; this should be done before the arsenic is applied and allowed to harden, when the application can be made and sealed without danger of either pressing on the pulp or forcing the arsenic out upon the gum. The tendency in the use of arsenic is not to leave it in contact with the pulp long enough. I think 36 or 48 hours is short enough even in

the most favorable cases, and yet if a very large quantity is used there is some danger of it being carried beyond the apex, especially in young teeth with large foramen, which means the ultimate loss of that tooth. After arsenic is removed it is best practice not to attempt removal of pulp, but to seal in tannic acid to harden the tissue or sodium hydroxid to partially saponify it. The practice of applying dialyzed iron to the pulp after arsenic is removed is both useless and dangerous, liable to cause serious discoloration of the tooth.

Either of the above preparations should be left sealed in for at least four days and ten is better. Before applying these, however, the pulp should be completely exposed, all decay removed and tested with a smooth broach. Many practitioners believe it best to remove the pulp immediately on the removal of the arsenic, and in many cases for lack of time and other special reasons this may be necessary. If, on the other hand, these other agents are used and allowed to remain until the pulp has been thrown off from the tissues beyond the apex, root canal cleaning is greatly simplified; especially do I like the use of solution of sodium hydroxid. I have used this preparation for several years, and in all cases where the pulp was completely destroyed by arsenic before applying it I am able to remove the pulp in one piece leaving the canals clean and white, a condition that is most desirable.

The cleaning of pulp chambers will be the subject of the next chapter, but before dismissing this subject I wish to refer to the treatment of arsenical poisoning of the gum tissue. When arsenic remains in contact with the gum tissue for any length of time wide destruction of that tissue results which often involves the periosteum, pericementum and bone.

By way of emphasis I will cite a case in point which came to me not long ago.

A lady who had formerly been a patient of mine went to a neighboring dentist, a young gentleman that she was somewhat interested in, and anxious to aid in building up a practice, and he devitalized the pulp of a superior second bicuspid tooth. She came to me and said: "Doctor, I want you to give me a few minutes time." She didn't tell me the dentist's name, and I didn't ask her, but she said that he had been six weeks attempting to devitalize that pulp, and that the tooth had gotten very sore and she was alarmed. Well, I put her in the chair and I examined her case, and I saw immediately the thing that had occurred. I applied the rubber dam that she might not know just what I was doing. I didn't want to tell her because she was very bitter against the young man by this time, and I didn't want her to know what had occurred there. I applied the rubber dam over the two teeth mesial and distal to the space where the trouble was, and then with a little manipulation I brought away,

I should say, a piece of bone fully a half by an eighth of an inch, or in other words, I brought away completely the entire alveolus between those two roots clear to the apex. I slipped it out over the gum, and threw it away, and she didn't see it at all and never knew what had occurred. I washed it out, and packed it with antiseptics and it is filling in nicely, but in all these cases where the gum septum peridental membrane and alveolar border are lost it is never fully restored and always will prove a source of annoyance and require constant watching.

If you get some arsenic on the gum accidentally, what is the thing to do? Swab it off with dialyzed iron, or what is better, freshly prepared solution sulphate of iron with magnesia. If at the second operation you find the gum is destroyed quite largely, what are you going to do? You may apply your dialyzed iron, but it won't do any good. You simply have to treat as you would a surgical wound. Remove the dead material, either by actual operation, or by cauterizing it with carbolic acid, nitrate of silver or something of that sort, and then keep the wound antiseptic, encouraging it to heal in every way you can. I shall speak of the management of these gums later in connection with another subject.

CHAPTER IX.

Cleansing and Filling Pulp Chambers.

The Pulp Chamber. Variations of the Form of Pulp Chambers. Removing Pulps. Filling Pulp Canals.

The Pulp Chamber.

The term pulp chamber is used to designate the central cavity in the dentine of the tooth, and is usually divided into a crown part known as the pulp chamber proper, and a root portion, known as the root canal or root canals. An accurate knowledge of the anatomy of pulp chambers is of the utmost importance, and without it many mistakes are made in operating, both in the preparation of cavities for fillings as well as in opening chambers for the purpose of removing pulp, treatment and filling canals. While pulp chambers often vary somewhat from the normal, yet an accurate knowledge of the normal will be most helpful in dealing with such abnormalities as may be met with in daily practice.

Teeth of different denominations have different canals, and it may be said that teeth of the same denomination often differ in this regard. In teeth with only one canal the pulp is usually conical, being large in the pulp chamber proper and gradually tapering to the apical foramen, which in fully developed teeth is very small. The following description and illustration is adapted from Black's "Dental Anatomy."

Fig. 23.

Longitudinal section through the center of the teeth, showing outline form of pulp chamber and root canals. (Adapted from Black.)

In the upper central and lateral incisors (Fig. 23, A, B, C, D) there is no distinct division of the pulp cavity into the pulp chamber and root canal; but there is one straight canal, from the interior of the body of the crown to the apex of the root, of which the crown portion is the larger. In young teeth, this has very distinctly the form of the surface of the tooth and root, except that it is much more slender. The largest diameter of the cavity is about level with the gingival line on the labial surface. From this point, the pulp chamber, or canal, extends toward the cutting edge of the tooth, about two-thirds the length of the crown, sometimes a little more, often less, and ends in a thin edge broad from mesial to distal. From the level of the gingival line towards the apex of the root it tapers very gradually and regularly to a narrow canal. Just within the apex of the root, almost at the end, there is usually a sudden contraction of the diameter of the canal, lessening it from one-third to one-half.

The pulp chamber and root canal of the upper cuspid (Fig. 23, e, f) is about the same in form as that of the central and lateral incisors, except that the coronal extremity has the central horn much extended toward the apex of the cusp of the tooth, and the mesial and lateral horns are practically absent. The coronal portion of the pulp chamber of the lower incisors is much flattened (Fig. 23, g, h).

At the level of the gingival line, the long diameter is from labial to lingual. The chamber extends towards the cutting edge of the tooth, about two-thirds the length of the crown, and in this extension its diameter is progressively diminished from labial to lingual, and extended from mesial to distal, following the contour of the surface of the tooth, and ends in a thin edge. In young teeth this has three short projections towards the mammelons on the cutting edges of the young, unworn teeth. The root has usually a narrow slit-like opening for the greater portion of its length, corresponding with the form of the flattened roots.

The pulp chamber and root canal of the lower cuspid (Fig. 23, i, j, k) are variable in size and form. At the neck of the tooth the chamber is usually irregularly flattened, with the longer diameter from labial to lingual, and the labial portion wider than the lingual. The coronal portion extends about two-thirds of the length of the crown towards the point of the cusp, ending in a point, or horn, which is often very slender. The form of the root portion of the canal depends on the form of the root. It is sometimes nearly round but more frequently it is sharply flattened for the greater portion of its length, becoming more rounded towards the apex.

Occasionally, this canal is divided for a part of the length of the root. In upper first bicuspids the pulp chamber and root canals differ

from those of the incisors and cuspids by a coronal chamber distinguished sharply from the root canals (Fig. 23, l, m).

The chamber is centrally located in the long axis of the crown of the tooth, the axial walls being about equal in thickness. The center of the pulp chamber is about level with the gingival line, or a little towards the occlusal surface. The occlusal walls are thicker than the axial, and vary in thickness from one-third to two-thirds of the length of the crown of the tooth. The form of the pulp corresponds closely with the form of the tooth. A horn extends from the coronal portions towards the apex of each cusp.

The root canals in upper first bicuspids that have two roots pass from the pulp chamber through the center of each root to the apex, and are known as the buccal and lingual root canals (Fig. 23, m). The buccal canal arises from the extreme buccal side of the pulp chamber, and the lingual canal from the extreme lingual side, and their course is almost parallel with the walls of these two portions of the pulp chamber.

The pulp chamber of the upper second bicuspid (Fig. 23 n, o) is similar to that of the first, but the horns of the pulp are usually shorter. In this tooth there is generally but a single root canal, and sometimes there are two canals which end in a common apical foramen, and sometimes these canals continue separately to the apex. The pulp chambers of the lower bicuspids (Fig. 23, p, q) seldom show a marked distinction from the root canals. There is, however, usually a coronal bulbous portion which connects with the pulp canal proper by an extended funnel-shaped construction. In the lower first bicuspid, the coronal extremity ends in a horn, which extends towards the point of the buccal cusp. The root canals of the lower bicuspids are usually large in the first half, tapering to a fine canal in the apical third of their length. The canal of the lower first bicuspid is usually nearly round, and that of the second is considerably flattened—and in both they are usually straight. Bifurcations of these canals are rare, but occur occasionally.

The pulp chamber of the upper molars (Fig. 23, R, S) is very distinct from the pulp canals, the latter often leaving the former by very small openings. The form of the pulp chamber is generally similar to that of the crown of the tooth; but the horns in the young tooth are often quite slender as compared with the cusps, and penetrate far towards the enamel. The length of these diminishes as age advances. In teeth much flattened mesio-distally, as often occurs in the upper first molars, and especially with the second, the equal thickness of the axial walls is usually maintained pretty closely, so that the flattening of the pulp chamber seems out of proportion to the form of the tooth. The floor of the pulp chamber is rounded or arched in the center (Fig. 24, A, f) and falls away

towards the mouths of the canals. The latter is situated in the portions of the angles of a triangle.

The opening into the lingual root is the simplest and most direct. Generally, it begins in a funnel-shaped opening inclining to the lingual,

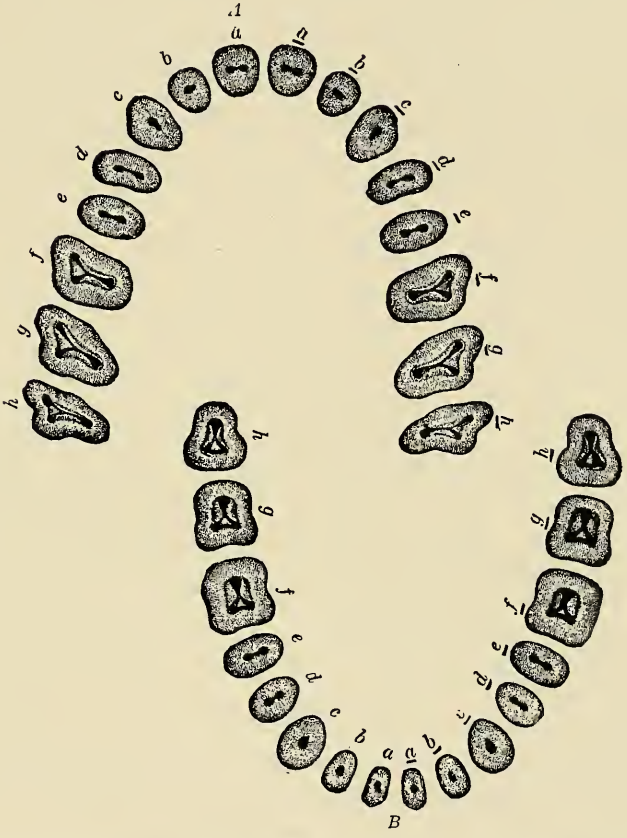

Fig. 24.

A, the upper teeth cross section showing the entrance to all the canals from *a* to *h* and *a* to *h*; *B*, the lower teeth cross section showing the entrance to all the canals with same letters.

which quickly narrows to the dimensions of a moderately small canal, and continues to taper to the apical foramen. It is usually straight, or but slightly curved.

The opening into the mesial canal is under the mesio-buccal cusp, close against the mesio-buccal angle of the pulp chamber. To find this canal the point of the broach should be directed into the mesio-buccal angle of the pulp chamber; and, while held against the wall within this angle, it is slid towards the root, and will rarely fail to glide into the

canal. The distal canal usually begins abruptly as a fine opening situated at the disto-buccal angle of the floor of the pulp chamber, so that a broach pressed into that angle will easily glide into it. But in some instances, especially in the upper second molars, the opening is in the floor of the pulp chamber at a little distance from the immediate angle towards the center of the floor, and then, in positions which limit the use of the eye, it is often difficult to find. In teeth much flattened at the neck, the opening of this canal may begin very close to the mouth of the mesial canal or close against the distal wall of the chamber, half-way from the buccal to the lingual wall or anywhere between this point and the disto-buccal angle. This description will do fairly well for all upper molars.

The pulp chamber of the lower molars (Fig. 23, T, W) has the same general form as the surface of the crown, but is generally rather more angular. The wall of the chamber towards the occlusal surface is convex toward the pulp; the horns extend from the extreme angles towards the apex of each cusp. The floor through the central portion is arched or convexed mesio-distally, and concave bucco-lingually (Fig. 24-Bf). The mesial wall of the cavity is flat and longer than the distal. The mesio-buccal and mesio-lingual angles are sharp and projecting, while the distal angles are rounded. The size of the chamber varies much. The root canals of the lower molars proceed from the mesial and distal portions of the pulp chamber. The mesial canal, at its mouth, is usually about as broad from buccal to lingual as the whole breadth of the chamber, including its angular projections. Either at or a little root-wise from the floor of the pulp chamber, it is usually divided into two very small canals which diverge at first, and approach each other afterwards, but usually remain distinct, each ending in its own apical foramen. Occasionally, however, they are united in the apical third of the root, and end in a common apical foramen. By placing the point of the broach through the mesio-buccal angle of the chamber and pushing it gently on, it will generally glide into the canal. The broach easily glides into the mesio-lingual canal by placing the point in the mesio-lingual angle of the pulp chamber and sliding it towards the root. The first inclination is to the mesial, but occasionally to the lingual, after which it curves to the distal and buccal. The distal canal is approached by a funnel-shaped opening, of which the central part of the distal wall of the pulp chamber becomes a portion. Its direction is a little to the distal, and generally very nearly straight to the apex. It is generally much larger than the canals of the mesial root, and is easily cleaned with the broach. If the mouth is wide open and the handle of the broach brought against the upper central incisors with the point directed against the posterior wall

of the pulp chamber, it will easily glide into the canal, and pass to the apical foramen. Fig. 24 should be carefuly studied; it represents the locations of all the pulp chambers.

Variations of the Form of Pulp Chambers.

Many variations of form occur in the pulp chambers and root canals. The roots of the teeth may be abnormally crooked, and then the canals will be abnormally crooked. In many instances the pulp chamber will have in it secondary formations, called nodules, which may be adherent to the walls or block the mouths of the canals and prevent a broach gliding into them. These also occur, occasionally, within the canals, partially blocking the way of the broach. Sometimes the pulp chamber will be filled with nodular deposits so completely that there seems to be no room for the tissue of the pulp. These deposits will have to be removed before the root canals can be reached and entered, after which the canals will generally be found open. These deposits occur within the pulp chambers of any of the teeth; but they cause annoyance more frequently in the molars. Occasionally lateral openings occur from the root canals to the surface of the root. I have seen more of these from the canals of the lower molars than from those of any other teeth. Generally they follow the course of the dental tubules, and open on the side of the root. They may diverge to one side and curve towards the apex of the root. These cannot often be detected, except in dissections of the root, and occur so rarely they may be ignored in practice. Sometimes the horn of the pulp approaches abnormally near the points of the cusps of some of the teeth, as in the upper first molar. Then the pulp is more liable to exposure in excavating carious cavities."

Removing Pulps.

Removing pulps is an operation that is sometimes difficult on account of the smallness, irregularity and inaccessibility of the pulp canals; particularly is this true in the buccal canals of upper molar and the mesial canals of lower molars—but in all single rooted teeth and the large straight canals the operation is very easy. The first requisite for such operations is proper instruments. A smooth fine piano wire broach, the Donaldson or Realization barbed broach of various sizes, and the Downey spiral broach of various sizes are the instruments most needed (see Fig. 25A). Each of these broaches should be carefully tested for weak places; particularly is this a necessity with all barbed broaches, for weak places are very liable to exist. The easiest way of making such a test is to take the handle and hold the point obliquely against the glass slab with enough force to spring it, at the same time rotating it; if a weak place

exists it will fracture at that point, and save you the annoyance of having it break in the canal. The next important thing is to secure the best possible access to the canals. I am amazed sometimes to see operators trying to remove a pulp from a three canaled molar through an opening in the central fissure the size of a No. 5 round bur. The complete removal of the roof of the pulp chamber is the best plan, which in most cases is easily done with an inverted cone bur. In opening up pulp chambers the operator should avoid disturbing the floor of the pulp chamber, particularly in molar teeth, for, as has already been alluded to, the floor is a guide to the entrance of the root canals and makes the finding of each a simple matter. I wish to deprecate the use of round burs for the purpose of opening pulp chambers, for with them the chamber floor is so liable to be cut and the entrance to the canals filled with fine chips, making it almost impossible to locate and enter a broach in them.

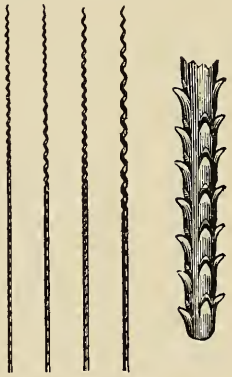

Fig. 25A.

All of this work must be done under the most thorough antiseptic precautions. The dam must always be in place, the field of operation cleansed and dried, the immediate tooth cavity sterilized and dried with alcohol, the broaches and other instruments used must be sterilized, and from the time you open the chamber nothing should ever enter except such instruments and agents as are placed there by the operator. After the chamber has been opened the cavity should again be flooded with alcohol to wash out all loose fragments and then dried with warm air; the drying has a tendency to shrink the pulp tissue, making its removal all the easier. The next step is to remove the large bulbous portion of the pulp with the pliers or a spoon excavator. In many cases where sodium hydroxid has previously been sealed, the entire pulp will come away with the pliers or excavator; indeed, I have several such specimens preserved that were taken from molar teeth in this manner.

A fine broach is then passed along the walls of the various canals to explore and locate any irregularities, after which a barbed broach is carried along the wall well into the canal and turned just sufficient to entangle the pulp, when it will easily come away in one piece; this will nearly always be true in large canals, but oftentimes canals are too small to admit of even the finest barbed broach with safety, for the danger of catching the barbs in the dentine is very great in small canals, with the result that a portion of the broach is left in the canal, which is a very difficult thing to remove. In these small canals I have been using for several years the Downey or Ivory twist of spiral broach. With a little experience they can be turned into almost the smallest canal, not only removing the pulp but enlarging the canal slightly. After the pulp is removed the walls of the canals should be scraped to remove the odontoblasts that may be clinging thereto. This can be done in all but the smallest canals with the barbed broach by introducing and withdrawing without turning or rotating it. It is considered best not to disturb these walls further than this in all except the very smallest canals; but in these small canals it is often necessary to enlarge them in order that they may be filled. The Downey broach is admirably adapted for this purpose, but the point needs to be dulled a little in order to prevent it boring through the side of the root in tortuous canals. This broach should not be turned into the apex before withdrawing and cleaning it, but it should be turned in and back, little by little, withdrawing and cleansing every few moments.

There still remains some very small inaccessible canals that cannot be cleansed in this manner, and for this several methods have been suggested.

First—The use of 50 per cent sulphuric acid to cut the soft tissue and dissolve a little of the dentine, thereby enlarging and cleaning the canal. It should be introduced on zephyr wool fiber with a platina iridium broach; except in rare instances it should not be allowed to remain in the canal but a few minutes, after which it is neutralized with a solution of sodium bicarbonate. This preparation is specially valuable in loosening pulp nodules and tumor-like deposits occurring sometimes in the canals.

Second—The use of pulp digestors such as carica papaya, dissolved in slightly acidulated water and allowed to remain sealed in the chamber for 10 to 20 days.

Third—Mummifying paste, for which I have no respect.

When the canals have been thus mechanically, and, when need be, chemically cleansed, they should be flooded with alcohol worked well down to the apex; this will wash up all loose particles; clean and dry the canals, in which condition they are ready for the filling. At this

point I want to emphasize the value of mechanically cleaning the canals rather than relying on medicines to do this work. This point was well emphasized by Dr. Logan in an article read before one of our societies recently.

Many operators advise the filling of root canals immediately after removing the pulp, and if the work of cleansing has been thoroughly done, with all precautions against infection, it would seem advisable after bathing in some mild antiseptic like my eucalyptol, oil cloves, trikresol mixture, and again drying with alcohol. This procedure is permissible in those cases where sodium hydrate has been applied following the arsenic, but in all other cases it is wisest to allow the dressing before alluded to to remain for a few days in order to be certain of our conditions before root filling.

Filling Pulp Canals.

There is no subject in operative dentistry that has received so much attention in the last 15 years as has the proper filling of pulp canals, and yet the ideal filling material has not been found. First came cotton, then wood, followed by gold, charcoal, tin, wool, chloride of zinc, paraffine, gutta-percha, and lastly balsam varnish—but none are ideal.

The ideal root filling should be non-porous, non-irritating, non-shrinking, and of such a nature that it can easily be placed. Such a material we have not, but the most nearly ideal material we have at the present time is gutta-percha, and is the one that is most universally used.

The technique of using this material for the filling of large canals is to first select a small piece of conical shape that by exploration you have decided will close the apical foramen. The dry and sterile canal is slightly moistened with eucalyptol, all excess removed and with the aid of a properly shaped root canal plugger the selected piece of gutta-percha is slightly heated and packed in the apical end, completing the operation by packing piece upon piece until the whole canal is tightly filled, using hot air to soften when needed.

In smaller canals it is customary to select a gutta-percha cone small and stiff enough to admit of being forced to the apex; the canal is, as before, slightly moistened with eucalyptol and chlora-percha—that is, gutta-percha dissolved in chloroform—is pumped into the canals with a fine smooth broach. In the very small canals much care must be exercised and the pumping continue for a considerable time to force the chlora-percha clear to the apex, then the selected cone is carried to place gently, allowing the excess chlora-percha to escape back into the cavity and not forced beyond the foramen. Where the canals are very small fine 14k. gold and also copper points are made to take the place of the gutta-percha. They are much stiffer and so can be forced where so small a gutta-

percha cone would not go. In the larger canals the walls only should be coated with chlora-percha and the gutta-percha cone placed. As the chloroform evaporates the gutta-percha should be warmed and again packed in order to fill in the shrinkage. Many use eucalypto-percha, others sandarac varnish instead of the chlora-percha. The object to be attained is to completely fill the canal and not force any material beyond the foramen. While this is a very delicate matter, a little practice will develop that intuitive perception that will enable one to do it well; the slight flinching of the patient is only valuable when we know by our sense of touch that it is the filling at the apex that is the cause and not air. It is impossible to accurately describe the filling of root canals. It is an operation which only the trained finger touch, as elsewhere in the field of operative dentistry, will make the successful operator.

CHAPTER X.

Suppuration of the Tooth Pulp.

Immunity and Susceptibility. Kinds of Pus. Fever. Symptoms of Fever. Suppuration of the Pulp. Cases of Open Cavities. Cases of Putrefaction Under Fillings. Treatment.

Suppuration.—The formation of pus; the act of becoming converted into pus, is the definition given, I think, by most authorities. Thus far we have for the most part been studying inflammation, which I have endeavored to show, when confined within certain limits, is purely a process of repair. When the healing process becomes infected with bacteria, then we have what is termed an infected inflammation, a condition unlike simple inflammation, which shows a tendency to confine itself to a local area and to heal; when it once becomes infected it shows a tendency to spread and take in the neighboring parts and no tendency to heal. In our study of inflammation of living tissue we stopped with the exudate of coagulable lymph into which tissue building cells congregate, and gradually transformation into granulation tissue, and then fibrous tissue and healing is completed by the growth of epithelium.

Let us return to our case at the point where tissue building cells are gathering in this coagulable exudate, and here introduce an element which often unexpectedly appears always uninvitedly, namely, certain micro-organisms. We see, then, a most interesting process, usually resulting in suppuration, but not always. Sometimes we will have the presence of pus forming bacteria in these inflammations in considerable numbers and no perceptible suppuration occurring. Why? This is a point I wish to clear up first.

First—The condition of the germ with which we infect.

Second—The condition of the whole cells in the part infected.

Third—The condition of the whole organism, constituting what is known as immunity.

Immunity and Susceptibility.

Immunity is the condition in which the body as a whole animal organism resists the entrance of disease producing germs, or, when they have entered, resists their growth and pathogenesis. The opposite of which is the term susceptibility, in which, instead of resistance, favorable conditions are present for the growth of these germs and their pathogenesis.

The study of immunity and susceptibility is perhaps the most interesting of all physiology and pathology.

Man suffers from many diseases which are never observed in the animal. The laity have always explained this fact by saying that animals are different from man; but the more the scientist contemplates this subject the more complex it becomes, and today the whole investigation is only in its infancy. It was early thought that the chemistry of the body constituents would explain all, and indeed this has a very important part in it; but it does not explain why, for example, the white mouse is especially susceptible to anthrax, while the house mouse is almost immune to its ravages. Nor is this the most remarkable thing about the subject. Why does one attack of yellow fever, smallpox or typhoid fever render the subject practically immune to the second attack?

And furthermore, a few drops of blood taken from the mouse recovered from tetanus injected into another renders it immune to that disease. These are some of the interesting things that we observe. I want you to get the various explanations for this thing, because if there is one subject a dentist should be familiar with, it is the subject of pus formation, how it occurs, what it does when it does occur, etc.

The first theory, perhaps, is known as the *Exhaustion Theory.* Pasteur explained this immunity by saying that the micro-organisms had used up all of some certain material in the body which is essential to the growth of these germs; hence they die from exhaustion, i. e., from lack of food. Hence the removal of this material by any means will permanently remove all liability to disease produced by these germs. Sternberg pointed out the weakness of this theory. He said if it were true we must have in our body a variety of this material, of smallpox, of measles, of scarlet fever, and a hundred others to be exhausted by its appropriate organism, and shows how exceedingly complex and stable the chemistry of the body must be in order to make this theory hold good.

The second theory is known as the Retention Theory. In the same year that Pasteur and Sternberg were working, Chauvan pointed out the fact that probably the progress of any disease may develop in the system a substance which hinders its further growth. There seems to be a large amount of truth in this; but if entirely true, what amount of material and how many different kinds would be added to our blood in case we had smallpox, measles, scarlet fever, typhoid fever and all the rest. Following this, in 1881, Carl Rosser showed the relation of phagocytosis to immunity. Similar observations were made by Sternberg in the United States and Koch in Germany. This theory was more thoroughly and fully developed by Metschinoff in 1884 advancing his theory regarding the process in a long series of experiments, he showed the relation of the leucocytes to bacteria. The phagocytes, which we have alluded to in a previous chapter, are cells without much resisting cell wall and are

capable of amoeboid movement. Outside of the body, if we place the amoeba in a suitable liquid containing bacteria although the amoeba possesses neither nervous system, eyes, nose, or volition of any kind, it will nevertheless seek out these bacteria. They surround the bacteria and really digest it completely. The property which enables it thus to find the bacteria I have explained under the head of chemotaxis. This is exactly what takes place in the body, and is what I have alluded to. Metschinoff, a man who has done perhaps more of real scientific work than any living man along this line, succeeded in catching some of these leucocytes, each containing an anthrax spore. After inoculating his subject with anthrax he succeeded in getting any number of these wandering leucocytes containing anthrax spores within themselves. He placed them in culture media, which, of course, was ill suited to the life of the leucocytes but better so to the bacteria, and watched the result. The leucocytes died and the germs lived and grew. Taking this, then, and the amoeboid movement, we have the process of suppuration and immunity as explained by Metschinoff, which is known as the *Metschinoff Theory of Phagocytosis.*

A word further in explanation of the action of leucocytes upon bacteria. Hankin and Hardy found three varieties of leucocytes had a part to play in the process, the outline of which is given in McFarland's work on bacteriology. Hankin and Hardy, taking up Metschinoff's suggestion, went to work to study just the kind of leucocytes that have to do in this thing. They found that certain eosenophilic cells approach and swallow up the bacteria; then certain other cells, known as the hyalin cells, take up the remains left by the former cells and destroy it. Another cell, which is known as the basophilic cell, supposed to be antidotal to the poisons surrounding the combatants, neutralizing the bacteria poisons and setting free the contestants.

We have another theory which I want to call your attention to, known as the humoral theory, a theory worked out by Buchner. In short, it is this, that the serum of the blood possesses certain germicidal powers which may be destroyed by heat, fever, etc. Jetter claimed this was due to certain salts contained in solution in this serum. Hankin thought this action was due to some substance contained in the eosenophilic cells. In no field has so much experimental work been done as in this. At the present time the relation of the blood serum is not exactly understood. The experimentation has given rise to the present theory of antitoxin. which, as I have previously stated, is at present in its infancy. In short, it is a process of cultivating germs in the living body, and taking the serum of this subject at a certain point when it contains poisonous products of these germs, and the tissue change, and injecting in man to im-

munize him. And this same process is now being developed to include immunity from infection. To what extent this theory will carry us the future alone can decide. Space will not permit me to go further into the subject. I simply wanted to bring enough of these theories to your attention to furnish a rational basis for what I have to say about suppuration.

Let us return to our consideration of suppuration, and perhaps this little apparent digression may aid us in comprehending what does occur. The cells, indeed the whole tissue involved in the process of repair, may possess sufficient vital force to withstand the onslaught of these micro-organisms, and they are literally destroyed and carried away, and we have no infection, which, of course, is Metschinoff's theory regarding non-infection. Every operator I know has had abundant experience to demonstrate the fact that simply introducing through the skin some infectious material does not always bring infection. I know that many of you have already pricked your fingers with broaches and exploring instruments that you knew were infected and had no result. Another time, you do not know how, you have pricked your finger, and the first thing you know you have a swollen finger, it begins to get sore and you have the whole process of inflammation and suppuration going on in it. Suppuration is a subject which has challenged the attention of the brightest minds among scientists for many hundred years, and the opinions of men have undergone most radical changes within the last thirty years, and, indeed, I may say that it is only within the last fifteen years that the cause of pus formation is at all understood. Up to that time the theory advanced by Bilroth, and afterwards elaborated upon by Conheim, seemed to satisfy most minds. This theory is briefly stated thus: Suppuration consists in an enormous multiplication of the cells of the part due to diapedesis of leucocytes, and that the fluid portion of the exudate fails to coagulate, and this with the softening of intercellular substance produces liquefaction of the forming tissue and pus. So far as it goes, it is correct, so far as we know. The only addition made since is to show exactly why the exudate fails to coagulate.

Bilroth's theory was that we have this great multiplication of cells out into the forming tissue. The serous exudate fails to coagulate, liquefaction of tissue forming cells and pus. All we have done to that in the last thirty years has been to try to clear up and explain why this exudate fails to coagulate.

Suppuration is a bacteriological process. The process by which this has been brought out, although exceedingly interesting, I can only hint at. Scientists generally agree with Bilroth and other experimenters as to what is seen in the suppurating tissue. Everyone agrees that what

Bilroth and Conheim and others saw at that time in the tissue was correct.

Lister, in 1869, threw some light as to how this liquefaction occurs. He called attention to the many micro-organisms found in the invaded tissue, as had also Pasteur, and suggested that perhaps they had something to do with the process. Lister probably was the first to clear up this subject at all. In proof of his claim that perhaps these micro-organisms had something to do with it, he set about to exclude bacteria from wounds and see what the result would be. This he started out to do in wounds made by the surgeon. It was just a little before this that the value of carbolic acid became understood as an aid in treating suppuration. Lister then operated under a carbolic and water spray. He first sprayed the whole room in which he was operating; the patient was cleansed; the part to be operated upon was thoroughly cleansed and sprayed, and after he had finished his operation the wound was covered with gauze and over this gauze cotton to keep the air away, and thus prevent the ingress of micro-organic life from the air. The world was surprised at his results. He had no suppuration. Up to this time the surgeon always looked forward to the time when this appearance of suppuration, or this sort of liquid or exudate would appear on the surface of the wound, or establishing of the secretions, and as the pus appeared he would look at it and examine the nature of it; under certain circumstances this is healthy pus, we will get healing right away here, believing that the suppuration was a necessary part of the process of healing. But Lister excluded all of these bacteria, operated under antiseptic conditions and he had no suppuration, but exactly what part the micro-organisms played in the process he did not know. He was ready to believe that micro-organisms had something to do with inflammation. He thought the degree of inflammation was somewhat dependent upon the number of these germs. This was away back in 1869. In 1881 he was still of the opinion that they were not a necessary part of pus formation in all cases.

So far as I can learn, Volkman was the first to declare that without micro-organisms we could have no pus. This was in 1881. A school was soon developed with Henter at its head, which had for its motto: "No pus without bacteria." This brought out most violent opposition.

Bilroth maintained that the bacteria were an accompaniment, not an essential part of suppuration. Pasteur was able to produce suppuration, with pus, in which he had destroyed all germs by heat of 110 C., leaving only the chemical products of those organisms. For the purpose of testing this idea severely men tried to produce suppuration with chemical irritants only—croton oil, turpentine, mercury, etc. Their results were

unsatisfactory, although many succeeded to their own satisfaction. A number were convinced that such a thing could be done, but the truth, doubtless, is that they infected their tests either from without or within the circulation, because they would succeed on one animal in producing pus and fail on two or three. The method was to make a fresh wound under aseptic precautions. A small glass phial charged with some concentrated irritant, as oil of mustard, was placed in the wound and the wound sewed over and allowed to heal. After the healing had occurred the flask was broken and the contents forced into the tissue. A hard swelling and severe inflammation was the result, but usually no pus. When pus did occur it doubtless was due to the presence of germs somewhere in the tissue or circulation, and carried there, a thing which occurs very frequently; hence surgeons will not perform a severe operation when there is a pus forming process going on somewhere else in the body. A thing that was noticed of course, in the cases where they succeeded in producing pus by chemical irritants, was that in this pus there were great colonies of bacteria, always. They were never able to get the pus without finding bacteria in it. Today it is fairly conceded among pathologists that we have no pus with micro-organisms. A word as to how micro-organisms do their work. Dr. Black says: "Pus formation consists in the fermentation and liquefaction of plastic exudate thrown out in the process of inflammation. It seems, therefore, necessary that we have inflammation and inflammatory exudate before we can have any pus formation." This inflammatory exudate is completely studded with leucocytes, as we have already explained, into this substance wander these micro-organisms. If the germs possess sufficient vital force and the condition of the exudate is so lowered as not to be able to resist them, they find lodgment and perform all the functions of life, and in this process a peptonizing ferment is formed, which in turn liquefies the exudate, little by little, and being filled with these reparative cells all is carried away in the form of pus.

Then suppuration takes place in the tissue by virtue of the peculiar peptonizing or digestive action which the bacteria exert upon the tissue. In the beginning the same changes occur as in inflammation. Some œdema of the part is first observed. At the same time leucocytes are accumulating, the intercellular substance is gradually undergoing transformation, and as you approach the point of pus formation there is an increasing number of leucocytes and some red blood cells with increasing liquefaction of this intercellular substance, and pyogenic germs abound.

As the virus acts more and more intensely on the part, the entire structure breaks down, being digested, as it were, by the chemical peptonizing substance, and the tissue liquefies and floats away a fluid pus instead of solid material.

This, then, is the process. Remember first we must have inflammation and the inflammatory exudate.

Second, we must have bacteria before we can have pus.

Kinds of Pus.

We have several varieties of pus, and it is subject to constant change depending somewhat upon the location and form of the disease as well as the condition of the patient. When pus is of a yellowish white color, and about the consistency of cream it is usually composed of a large number of pus globules, and is known as healthy or laudable pus. You have observed when you have abscesses filled with this kind of pus, that when you once evacuate the abscess and disinfect the part you have recovery immediately.

It is the sort of pus from which recovery is readily made.

When pus is thin and reddish and streaked with blood it is called sanious pus, and very frequently is mixed with particles of fibrin and dead tissue. This sort of pus is mostly seen in certain bone diseases. Whenever you have caries of the maxillary bones you will always have this kind of pus and in phagedenic ulcers abscesses that have been standing sort of dormant for years, and tumors, etc. On opening into a tooth whose pulp is dead, the kind of pus that comes away will determine somewhat the condition beyond the apex. If you have the yellowish white pus you have a condition not to be dreaded very much, but when thin reddish pus comes sweeping down through the cavity you have a condition affecting the bone, almost always, and a condition that is going to be slow to heal.

Then watery, acrid pus is termed ichorous pus, and is common to chronic ulcers and certain bone diseases. When coming from the mucous membrane pus is called muco-pus. From the serous membrane, sero-pus.

Thick, ropy pus of syphilitic abscesses is termed gummy pus. When the sanious pus contains flakes of coagulated fibrin it is called cheesy pus. It is a sort of pus that very frequently is seen in the bone abscesses in abscesses of the superior maxillary bone, for instance, wherever pus is sort of confined under pressure, you will get pieces of white coagulated fibrin in the pus.

In the suppurative process we usually have as an accompaniment, fever. It is usually an accompaniment of inflammation and suppuration. Indeed, it always occurs in acute suppuration. Whenever you have an acute alveolar abscess forming, if it be any way violent in nature, if it has developed very rapidly, you will always have the patient presenting fever, as indicated by your thermometer.

Fever.

What is fever? An abnormal elevation of the body temperature. In hyperemia or simple inflammatory conditions we have a rising temperature in the part due to increased oxydization on account of the increased blood cells, but this is not fever, strictly speaking. Only when we have a rise in the whole body temperature do we regard it as fever. In ordinary acute alveolar abscess we sometimes have a rise in temperature to 101, up to 105 in severe cases. I want, if I can, to impress upon you the value of using this, as a diagnostic aid.

Often patients will present suffering severe pain. Often they will present with a tooth very sore and you wonder whether or not the process of suppuration has started in or whether you simply have a case of apical pericementitis. If you will take your thermometer and put it under the tongue and find a rising temperature to 101, you can make up your mind that the patient is being poisoned with the pus. This fever is the result of the poisonous products of micro-organic life. To understand it thoroughly a knowledge of the laws governing the mechanism of heat production and heat dissipation is essential. The normal temperature in a state of health is 98 4-10,° or 37 C., which is practically stable, varying slightly in torrid and frigid zones. The body is constantly producing heat by the process of combustion, oxygen being taken up by the tissue and carbonic acid eliminated. Enough heat is produced daily to raise the body temperature forty-eight degrees Centigrade, an amount far beyond possible life. Then there must be an arrangement by which this heat is liberated and the whole process is carefully balanced in order that the temperature remain stable. If we take a large amount of food, or unusual exercise, an increased amount of heat is produced; during sleep or repose the amount is decreased. The red face, moist skin, increased respiration, and all are evidences of this regulating process at work. The increased amount of heat production is offset by the increased amount of blood in the surface being cooled. You must remember that the temperature lowers slightly as we get away from the center of the organism. The surfaces of extremities are normally cooler than the thorax contents. The extra heat produced by food is rapidly carried to the surface to supply the tissue and be cooled. The point I want to bring out is that there is an automatic arrangement which seems to protect the body from ordinary changes to which it is subjected. The mechanism or arrangement only works within certain limits. All heat produced in the tissue is produced by assimilation of nitrogenous material brought about through the nervous and partly by the muscular tissue, and is a chemical process in which oxygen is absorbed and carbonic acid given off. Anything that will increase the amount of heat produced and at the same time interfere with

its dissipation, will produce fever. When a patient gets a temperature of 105 the nurse immediately understands that she must bathe the body in cold water, the philosophy of which is simply the cooling of the blood through the surface. So long as fever does not endanger the burning up of tissue it is not a dangerous thing, consequently it is not the habit of controlling fever by systematic medication in typhoid fever, and such, but by the constant cooling of the surface. Indeed, in typhoid fever many cases are bathed ten and a dozen times in twenty-four hours with cold water.

Symptoms of Fever.

The early symptoms of fever is a sense of lassitude or malaise, and if you examine the patient you will find there is a slight rise in temperature and rapidity of the pulse. The skin of the head and body feels warm to the touch, although the extremities may be cold. If the attack is severe and the temperature rises rapidly this condition is followed immediately by what is known as a chill. The skin is cold, particularly in the extremities; usually looks pale or slightly purple, accompanied with involuntary chattering. This will last for an hour or two, and is quickly followed by a sense of heat, flushed face. During the chill the patient crouches over the fire and wants to be covered with blankets, etc., when the chill passes they want the clothing removed again. If the fever be due to pus production, or rather if pus poisoning is gradual, there will usually be an absence of chill with but little fever. In all acute suppuration we have a decided rise in temperature, as I have stated, 101, 102, 103, 104, 105 or even 106 occasionally from acute alveolar abscess.

The thermometer is a diagnostic aid in these cases. If there is a chronic case where a large amount of pus is present we will also have some fever. What is it that causes the fever?

Just how are these symptoms brought about? We stated that suppuration was purely a bacteriological process.

Can the same be said of fever? Not all kinds of fever are due to micro-organism action directly, but all pyogenic fever is. It is probably an effort of Nature to rid herself of some irritant which may be the product of bacterial action or of the bacteria themselves. This irritant may be in the nature of a ferment-like substance, or in some cases may be the result of cell disintegration. Bacteria do not grow well except in nearly normal temperature, and it may be that this rise of temperature has a beneficial result in inhibiting the growth of these low forms of life within the body. Indeed, this is the late idea regarding fever, that it is simply an effort on the part of Nature largely, to throw off these bacteria. Warren says—"In general, it may be said that fever is due to the presence

in the blood of a pyogenic substance of an organic nature that may have been produced by bacteria. That is the most common way. Second, to the presence of bacteria, or finally, to some ferment-like substance which has resulted from cell disintegration."

The poisonous products of bacteria, known as leucomaines and tox-albumens, act directly upon the nerve centers in such a way as to interfere with the mechanism of heat production and heat dissipation. Sufficient has been said regarding suppuration in general to enable the reader to follow the process as seen in the tooth pulp.

Suppuration of the Pulp.

Putrescent Pulps.

In the chapter on inflammation of the pulp we endeavored to show that inflammation sometimes only affects small areas around the point of exposure. When affected in this area, by some of the active pus germs, rapid liquefaction of the exudate and pus is the result. If there be a ready way of escape through the horn of the pulp out into the cavity, this process may be slow and the main body of the pulp may remain alive for some time, and death and suppuration come gradually, little by little, painlessly. But if that exit is closed, or nearly so, a rapid increase of inflammation will soon involve the whole pulp tissue, perhaps within eighteen hours, and on to rapid infection and suppuration of the whole organ. This is usually painful process, lasting from twelve to twenty-four hours, and sometimes, unless relieved runs directly into alveolar abscess. Such a process as this is usually what occurs when we cap an infected pulp or cover in some infective material. These cases, after once started usually work rapidly, and pain ensues and continues say about twenty-four hours, and with a sudden stop the pulp is all dead, and may be it will pass on to the peridental membrane and the tooth becomes sore to the touch, and alveolar abscess almost certainly will follow, although occasionally in robust individuals, persons whose circulation and elimination are good, it may stop for a time and all soreness pass away. The first or chronic form of suppuration, where progress is slow, little by little, is the most common. When the pulp once starts to suppurate it rarely recovers. The tissue cannot heal by cicatrization, i. e., it does not cover over its injury with epithelium as other wounds do, therefore it is always liable to reinfection in case the tissue does throw off one attack. When you open into these cases, say when simply the horn of the pulp is suppurated, you at first decide that you have a dead pulp to deal with, but when you attempt to pass your fine smooth broach through the opening up into the canal, you suddenly produce pain; you are then surprised to learn that a little way down the pulp is alive and normally sensitive. Oftentimes

it is fairly healthy; the living vital portion has separated itself from the suppurating portion by this wall of plastic exudate, similar to all inflammatory processes.

That is the usual way in which pulps die from infection. It sometimes happens that the pulp tissue in one canal in a three-rooted tooth will remain alive while the other two are undergoing suppuration. I think all have probably seen cases where a typical acute alveolar abscess forming, upon opening the tooth two canals probably filled with suppurating material, and the other canal sensitive, unable to enter it at all. Then, again, the suppuration may pass along down the center of the tissue, as shown in Fig. 25.

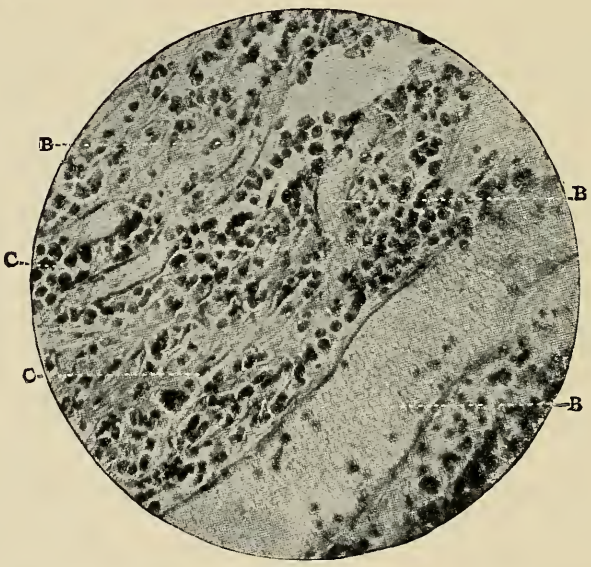

Fig. 25.

Chronic inflammation of the pulp. B, blood vessels crowded with corpuscles; C, nuclei of inflammatory cells. (Hopewell-Smith.)

Pus will work its way down, following the direction of the blood vessels until the center is largely destroyed and suppurated, and yet along the wall next to the odontoblasts the tissue is alive. In other of these cases there may have been symptoms of hyperemia, toothache a little while at a time, then passing away completely. Patients will say that perhaps six months or a year ago they had a severe toothache in a tooth; it was very sensitive to thermal changes. By and by it all passed away, and the tooth is comfortable. No matter what the symptoms are in these cases, you must rely on what you see while making your excavation and exposing the pulp, rather than outward symptoms; the latter are often

misleading. I repeat here for emphasis pulps inflame, suppurate and die in this slow chronic way without causing the patient the slightest annoyance.

Cases of Open Cavities.

"When the pulp of a tooth is exposed and becomes the seat of that series of vascular and nutritive disturbances—hyperaemia, inflammation and suppuration—eventuating in its gradual death, the necrotic portions undergo putrefactive decomposition. Several processes are in operation at the same time, so that different portions of the pulp exhibit differences in chemical composition, differences in the nature of the infection and also in the pathological conditions existing.

For example, while the apical portion of the pulp is the seat of inflammation and suppuration, the portion of the pulp previously destroyed through these processes, is the seat of later stages of chemical destruction, until that portion which was first acted upon is being revolved into the end-products of albuminous decomposition, of putrefaction.

In this serial decomposition albuminous substances are first transformed into peptones and allied substances, some of them being very toxic. Compound ammonias, known as ptomaines, or animal alkaroids, are probably next formed. Next the nitrogenous bases-leucin, tyrosin and the amins (methyl, ethyl, and propyl) make their appearance together with organic fatty acids. Next aromatic products, indol, phenol, creasol, etc., and finally hydrogen sulfid, ammonia, carbon dioxid, and water.

By alternating processes of hydration, reduction, and oxidation, bodies of increasing simplicity of chemical composition are formed. Miller found in the deepest portions of the degeneration, putrefying pulps, where inflammation and suppuration were in progress, a preponderance of small cocci and diplocci, and proceeding toward the open pulp chamber an increasing number of large cocci, several forms of bacilli, vibrios, and other spirillae, spirochaetae, and long thread forms.

Until infection of the pericementum occurs these cases give rise to no symptoms, except odor.

Cases of Putrefaction Under Fillings.

When a filling is placed over an infected pulp, or when the pulp dies subsequent to the insertion of the filling, the organ undergoes decomposition, the decomposition being carried on in this instance with the access of air, i. e., is accomplished by anaerobic organisms.

Miller found that bacteria of pulp-putrefaction cultivated in gelatin, with and without the access of air, exhibited a difference in the poisonous properties of their products. Those developed with free access of air produced reaction and more extensive suppuration than those developed without the access of air."—Kirk.

Treatment.

What shall be done for these cases?

Should they ever be capped? No, for they will all die.

Shall we proceed to devitalize at once? No, not until the suppuration has been stopped—the reason therefor relates to the fact that the presence of dried suppuration material in the chapter will prevent the arsenic from coming in contact with the living portion—and also there is some danger of setting up severe pain from sealing an active suppurative condition without proper drainage. It is not considered good practice to attempt pressure anesthesia in these cases. There is great danger of forcing the infective material beyond the apex and causing a most violent acute abscess. It is in this class of cases where the pulp is partially dead that one is tempted to use cocain, but in my hands these cases are unfavorable.

The treatment should be as follows, of course varying somewhat to meet the various conditions presented where the pulp is completely dead and undergoing suppuration, the first steps are the same as where a portion of that organ remains alive.

The rubber dam should be applied and the field sterilized, cavity washed, disinfected, and the pulpal wall removed as thoroughly as possible avoiding pressure on the contents of the chamber.

I find the inverted cone bur very helpful for this purpose.

The next step is to absorb away with cotton any exuding pus and wash with alcohol and dry thoroughly then seal in a good antiseptic such as beechwood creosote, 1-2-3, or camphophenique, trikresol, oil cloves, etc., which should be allowed to remain from 24 to 48 hours. At the next sitting if all has been quiet and no pus present arsenic method of destroying vital portion may be followed. In case the entire pulp is dead, and suppurating it can usually be thoroughly mechanically cleaned at the second sitting, and one of the agents suggested sealed in for another 48 hours, when the canals should be ready for filling. In managing these cases it is well not to poke around in the canals with a broach very much until the contents have been disinfected.

If this outline is carefully followed and no infection material forced beyond the apex the treatment of putrescent pulp is a very simple matter.

CHAPTER XI.

The Bacteria of Pus.

E. S. Willard, D.D.S.

Constantly present in the atmosphere, in water and in the dust; in the mouth, on the skin and inhabiting the air-tracts of the human body, are to be found micro-organisms, which, for the most part, are perfectly harmless. Some, however, will prove to be pathogenic when a lesion or break in the mucous membrane or skin (which act as outer defenses) affords them entrance into the tissues, or when some other abnormal condition favors their multiplication.

Of these micro-organisms I wish to speak more particularly of a certain few which seem to exhibit pathogenic characteristics only by their ability to form pus, and for this reason are known as Pyogenic Bacteria. There are other forms, which, under certain circumstances produce pus, but are not classified as pus producing micro-organisms, because they are more strictly identified with the diseases that they are known to cause.

The character of the pus produced by one organism does not differ from that produced by another organism. In fact the character of non-specific pus, or pus that may be produced by some powerful irritant as croton oil or carbolic acid, does not differ from specific pus or that produced by living germs, for in each case the cause is chemical, micro-organisms producing pus by the chemical operation of their enzymes or digestive exudates. The color, odor, or quantity of pus, however, are controlled by the particular species growing in the abscess.

It is a fact known to bacteriologists, that where disease is present as the cause of micro-organisms, the natural flora of that particular locality is in some cases, entirely absent, while the specific germ of the disease dominates.

In suppurative lesions, while there are many cases of contamination of species, yet quite frequent are the instances where pure cultures are obtainable from the inoculation of culture media with pus, even from localities that normally are teeming with varied forms.

Some of the organisms known to produce suppurative conditions are as follows:

Staphylococcus Pyogenes Aureus.

Staphylococcus Pyogenes Citreus.

Staphylococcus Pyogenes Albus.
Streptococcus Pyogenes.
These are known as the pus cocci.
Micrococus Tetragenus.
Pneumococcus or Diplococcus Lanceolatus.
Gonococcus.
Bacillus Pyocyaneus.
Bacillus Typhosus.
Bacillus Coli Communis.
Streptothrix Actinomyces.

Many other forms, among them some of the yeasts and moulds, might be mentioned, but this will suffice, and from these I will select three for description which are, strictly speaking, pus producers.

Name. Staphylococcus Pyogenes Aureus. Meaning the golden pus producing staphylococcus. Fig. 26.

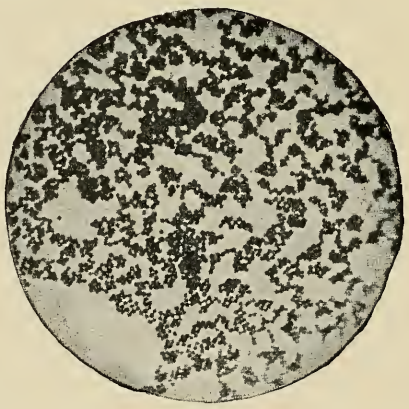

Fig. 26.
Staphylococcus pyogenes aureus from agar culture. (McFarland.)

Morphology. Spherical. It multiples on two or more poles irregularly taking a growth form, as its name signifies, after the manner of a bunch of grapes; groups of irregular dimensions, or it may be found as single cells or as diplococci. It measures ordinarily about .8 microns or about 1/10 the diameter of a red blood corpuscle i. e., 1/31000 of an inch; and it has been estimated that one grain of these individuals will number something like 125,000,000,000. A little figuring will show that a space measuring about 1 cu. millimeter could contain 1,952,625,000 or nearly 2,000,000,000 cells.

Discovered by Rosenbach in 1884.

Origin. It is the most common of the pus producing organisms and variously estimated as present in from 50 to 80 per cent of the

abscesses examined. It might be said, however, that in abscesses of the mouth the white variety of the plant is more frequently met with; the staphylococcus pyogenes albus. It is found in saliva, on the skin, in water, on particles of dust floating in the air or wherever dust may settle.

Mobility. It has no motion, having no flagella.

Spores. None. It multiplies by fission.

Staining. It is easily stained by all the common table stains, also by gram.

Growth. It grows very readily on all the ordinary culture media.

Buillon shows a cloudy appearance with yellowish sediment and a decided acid reaction.

Gelatin stab culture shows decided liquefaction along the entire tract of the needle. The growth shows a yellow precipitate at the bottom of the liquefaction.

Agar slant culture shows a moist golden yellow streak on the surface of the media.

From all of these cultures a peculiar sour or acid odor is noticeable.

Aerobiosis. It grows best in the presence of oxygen, where it produces its color, but will grow as a facultative anaerobe, and as such, produces no pigment. The optimum temperature is about 37 Centigrade, or that of the incubator, though it will grow at ordinary or room temperature.

Pathogenesis. The staphylococcus aureus is ordinarily a parasite though it will grow as a saphrophyte as is seen from its growth on ordinary culture media. Its enzyme separated from the media will produce characteristic pathogenic results in the formation of pus in living tissue. This organism is particularly pathogenic to man, and external applications of pure cultures have been known to produce suppuration and carbuncles (Garre), while inoculations may result in pyemia, infection of the kidneys, or metastatic abscesses. The infection, however, is more inclined to be local and less violent than that produced by the streptococcus pyogenes, and is, therefore, not very serious. Sterilization may be easily effected by subjection to streaming steam or the application of various germicides. Sterilized cultures contain the poisonous productions of the germs, and will produce suppuration.

Diagnosis. Microscopic observations will oftentimes lead to a suspicion of the presence of the staphylococcus aureus in suppurative areas, and microscopic examination of the pus with artificial cultivation will reveal the characteristics as above described.

The staphylococcus pyogenes citreus and the staphylococcus pyogenes albus are considered by some to be the same as the staphylococcus

pyogenes aureus, except that they grow under different conditions. That they are of the same species there seems to be no doubt, for all that may be said of the aureus may be said of the others, unless it be in so far as color and virulence are concerned; the albus being white and the citreus producing a definite lemon yellow pigment: Again it is the common opinion that the staphylococcus aureus is more virulent than the citreus and the citreus more virulent than the albus.

Name. Streptococcus pyogenes, this of all the pus producing organisms is the most dreaded by the surgeon. Fig 27.

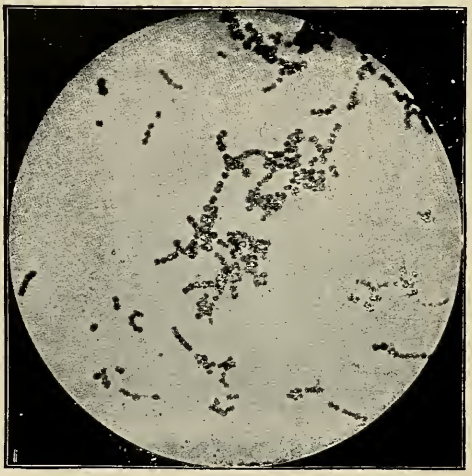

Fig. 27.
Streptococcus longus from a fatal case of pyemia. Magnified 1,000 times. (Hopewell Smith.)

Morphology. It is in many respects similar and in other respects quite unlike the staphylococci just described. It is spherical in form. As the name implies, it grows in chains, multiplying upon one pole. These chains may be of greater or less length. Names have been given to supposed varied species of Streptococci according as they are known to assume greater or less length of chain, as Continuousum, longus, brevis, media, etc. Whether these are varieties of the same or of different species is yet a question; it is, however, certain that they seem to exhibit different physiological phenomena and show greater differences than may be noted between the staphylococcus aureus, citreus, and albus. Some are not known to produce pus. The particular form under discussion distinguished as pyogenes usually extends to from 6 to 8 cells in length, though oftentimes in bouillon the extension will be to upwards of a hundred in number. In size the cells are a trifle smaller than the staphylococcus.

Discovered by Rosenbach in 1884.

Origin. In abscesses, pyemia, erysipelas. Found in the mouth, nose and throat. It seems to find its natural habitat about or in the vicinity of human beings.

Spores. None have been discovered. It multiplies by fission.

Stains by all the common table stains and by gram.

Growth is readily obtained on a slightly alkaline medium, though its vegetative function does not seem to be so well marked as in the staphylococcus.

Bouillon growth in the incubator shows at times a clouded medium with white precipitate, again the medium will be clear with the white precipitate at the bottom of the tube, and also attached along the sides.

Gelatin. We note that it does not liquefy, but small white colonies are formed along the entire track of the needle in stab cultures.

Agar slant culture. The growth is very slight, looks like small white drops and does not tend to run over the surface.

Aerobiosis. Grows best in the presence of oxygen. It is a facultative anaerobe. Grows at 37 Centigrade and gives a feeble growth at lower temperatures.

Pathogenesis. This organism seems to be more particularly a human parasite. It is pathogenic to man, rabbits and mice, and its virulence is particularly marked, producing spreading inflammatory infection, septicemia, pyemia and erysipelas, in which the organisms seem to infest the lymph channels. Pure cultures of streptococcus pyogenes may at any time be made from the pustules of erysipelas sores. Most of the more serious suppurative conditions of man are due to the presence of this organism.

Diagnosis. Microscopic observation of suspected pus, as in empyemia of the antrum. Artificial cultivation on glycerine agar in the incubator and intravenous inoculation of rabbits.

Name. Bacillus pyocyaneus. The micro-organism of green pus. Fig. 28.

Morphology. A rod form, small in size, scarcely exceeding 1 micron in length by from .3 to .5 microns in width. The ends are rounded. It is sometimes found in short chains but nearly always detached or as single cells.

Discovered by Gessard in 1882.

Origin. It is very commonly met with in nature. It is found in the air on particles of dust. It is found all over the body, and in the internal organs of man and animals. Pus, when infected with this organism or caused by this organism, becomes green in the presence of oxygen.

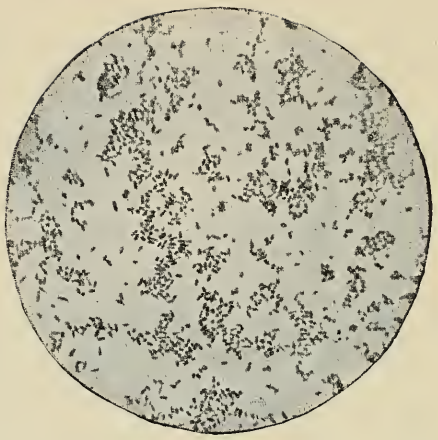

Fig. 28.
Bacillus pyocyaneus from an agar culture. (McFarland.)

Motility. Active. It has one flagellum.

Spores have not been discovered. Reproduction is by fission.

Stains by all the common anilin dyes and by gram.

Growth is readily obtained on artificial media.

Bouillon soon becomes cloudy and a white growth soon forms on the surface. Very early in the growth the upper part of the medium becomes green, and gradually extending deeper, it finally becomes brown.

Gelatin stab cultures. Liquefaction occurs along the track of the inoculating needle. A white growth shows upon the surface. And as in the case of bouillon, the green pigment shows at the top of the gelatin, gradually extending downward and finally becoming brown.

Agar slant shows yellowish or whitish slimy growth along the line of the inoculation and the pigment production gives the same indications as in gelatin.

Potato shows a brownish slimy growth; oftentimes no green appears.

Aerobiosis. Facultative anaerobe. The organism seems to grow as well out of the incubator as in it.

Gas production. It gives off different gases, and has a characteristic aromatic odor.

Chromogenesis. Two pigments are developed, a dark brown, or pyocyanin, and a beautiful deep green, or fluorescin.

Pathogenesis. This organism shows peculiar phenomena; at times it is harmless, and again decidedly toxic. Subcutaneous injections in guinea pigs produce violent inflammation and death. Post-mortem examination shows bacteria. Animals infected with anthrax, after being inoculated with the bacillus pyocyaneus, have been known to recover.

Bacillus tetanus, known to be an obligate anaerobe, will when contaminated with the bacillus pyocyaneus, grow as an aerobe. This may account for many deaths by lockjaw.

Diagnosis. It is most often discovered by the green pigment which it elaborates, but must be distinguished from the bacillus fluorescens by its liquefaction of gelatin in stab cultures.

CHAPTER XII.

Diseases Affecting the Peridental Membrane About the Apices of the Roots of Teeth.

Histological Structures of the Peridental Membrane. Functions. Structures. Cells. Blood Supply Nerves. Apical Pericementitis. Causes. Symptom. Treatment. Chronic Apical Pericementitis. Cases. Treatment. Alveolar Abscess. Causes. Symptoms and Pathology. Treatment. Chronic Alveolar Abscess. Aneurysm. Blind Abscess. Treatment of Pulpless Teeth. Special Cases.

Histological Structures of the Peridental Membrane.

Before considering the pathological conditions affecting this membrane it seems wise to briefly review its histological structures.

The peridental membrane is the soft tissue occupying the space between the tooth root and the alveolar wall. In the literature it is often referred to as dental periosteum, pericementum, alveo-dental periosteum.

It completely surrounds the tooth root from the enamel line, and serves as a connection between the tooth and its bony socket as well as gum tissue. It is thickest in childhood and thinnest in old age.

Functions.

This membrane differs from true periosteum in that both its surfaces are functioning. It can be said to have three functions, first, a physical function, that is it serves to maintain the tooth in its socket and to hold the gum around the tooth neck. Second—A vital function. The building of bone on the alveolar wall and of cementum on the tooth and it always maintains the tooth vitality after the pulp has been destroyed. Third—A sensory function. It not only transmits pain sensations, but the entire sense of touch is supplied by this membrane.

Structures.

The peridental membrane is made up of dense fibrous tissue, and in addition has certain cells, blood vessels and nerves. The arrangement of the fibres is intended to hold the tooth and support it against force from every direction.

For convenience of description, histologists divide the membrane into three portions, first, the gingival portion which includes all the membrane just under the free margin of the gum and over the border of the alveolus; second, the alveolar portion, which includes all the membrane from the alveolar border to the root apex; third, the apical portion which surrounds the immediate root apex and occupies what is known as the apical space.

If we examine the gingival portion under the microscope beginning at the center of the mesial or distal we will observe the fibres at the enamel junction of the cementum passing out from the cementum at right

angles to the long axis of the tooth, gradually dipping down to unite with the periosteum of the alveolus and some of the fibres pass directly into the gum septum and others pass over the border of the alveolus to mingle with the fibres of the adjoining tooth, and some of them passing directly into the cementum of that tooth, sometimes called dental ligament. Fig. 29.

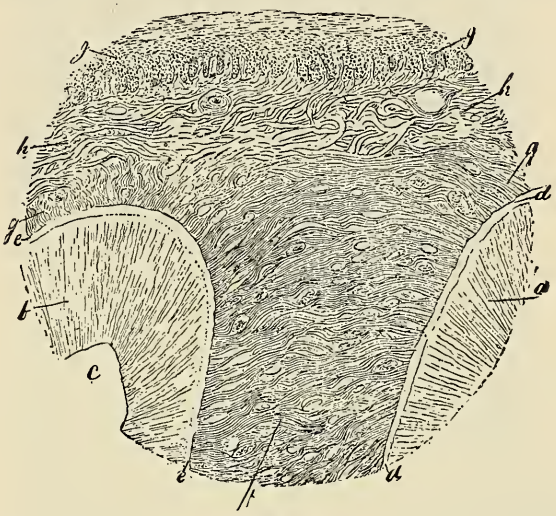

Fig. 29.

Cross section of central and lateral incisors below the rim of the alveolus through the neck of the teeth. *A*, central; *b*, lateral; *c*, pulp chamber of lateral; *d*, *d*, cementum; *e*, *e*, cementum; *g*, *g*, fibres of peridental membrane; *h*, *h*, *j*, *j*, epithelium. (Black.)

This you see is intended to hold the gum septum in position between the teeth and hold the tooth to the alveolar border as well as to the neighboring tooth. Passing around to the mesio-lingual or mesio-labial you will see the fibres pass out and turn to the right and left on a tangent entering the periosteum and gum (see Fig. 30). These are intended to prevent the tooth from rotating.

On the labial and lingual the fibres pass out at right angles directly into gum and periosteum and bone. In the alveolar portions the fibres coming out of the cementum in its occlusal portion pass at right angles into the alveolus, while a little nearer the apex they incline occlusally, and still a little nearer they incline still more.

In the apical portion the fibres are arranged somewhat fan-shaped over the apex. These fibres often pass out of the cementum in little bundles and split up before entering the alveolus. In examining the cementum it is seen that these fibres do not all penetrate to the first layer—that is, the layer immediately over the dentine—some pass in, only the last layer.

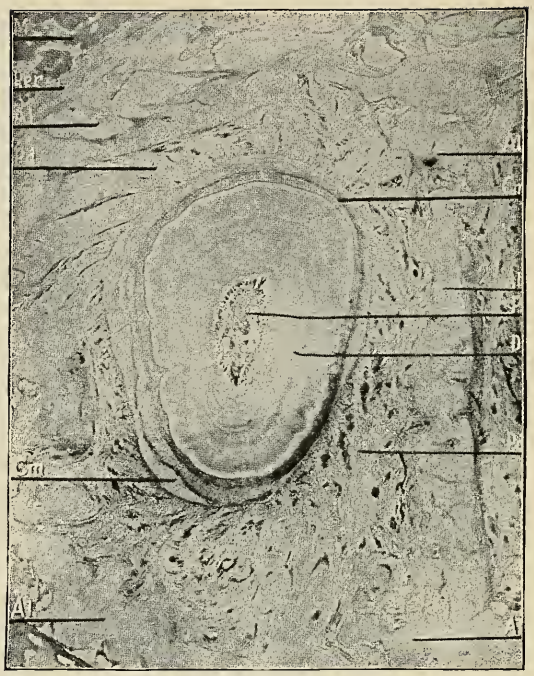

Fig. 30.

Transverse section of peridental membrane in alveolar portion. M, muscle fibres, periosteum; Al, bone of alveolar process; Pd, peridental membrane; Cm, cementum; P, pulp; D, dentine. (Noyes.)

Cells.

The cells of the peridental membrane are—I, Fibroblasts, spindle shaped cells whose functions seem to be the building of membrane fibres. II. The cementoblasts which lie in around the fibres on the cementum side; their function is the building of cementum (Fig. 30). III, Osteoblasts, which lie in around the fibres on the bone side; their function is bone building. IV, Osteoclasts, oftener called myaloplaques or giant cells. They are not always present, but seem to appear at times around among the fibres from some cause. They are the bone, cementum and dentine destroyers; when active they lie down close to the bone or tooth. They sometimes seem to be active when no cause can be assigned. Often cementum and even considerable dentine is cut away and again filled with cementum (Fig. 31). V, Epithelial cells. There is a set of cells that resemble epithelial cells which group themselves together so as to appear as epithelial glands (Fig. 32).

Many dispute their presence and think what is seen is something else, but the strongest evidence of their nature is the fact that the peridental

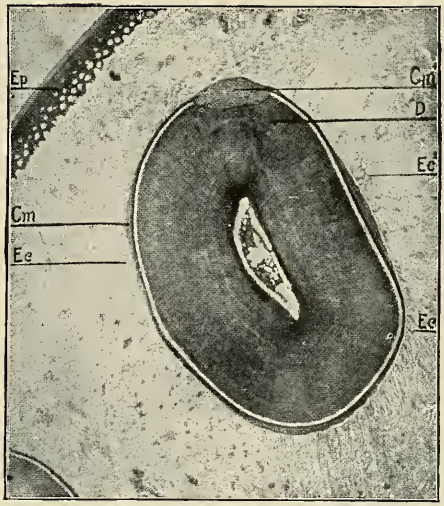

Fig. 31.

Transverse section of peridental membrane in gingival portion. *Ep*, epithelium; *Cm*, cementum; *Cm*², cementum refilling absorption; *Ec*, epithelial cords or glands. (Noyes.)

membrane has a secretive function. Many substances taken into the system are excreted around the gum margins.

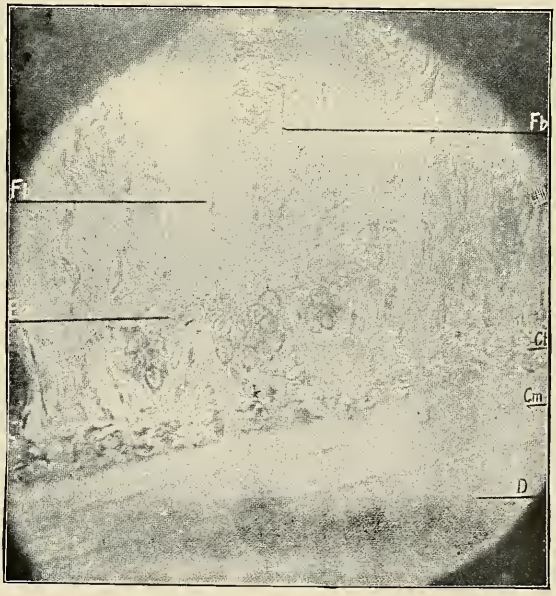

Fig. 32.

Peridental membrane next to cementum highly magnified. *Fb*, fibroblasts; *Cb*, cementoblasts; *Cm*, cementum; *D*, dentine; *Ec*, epithelial cords or glands. (Noyes.)

Blood Supply Nerves.

The blood supply of the peridental membrane is very abundant. Blood vessels not only enter at the apex, but from the bone and also over the alveolar border, and are distributed everywhere throughout the tissue twining in and out among the fibres. The capillaries are believed to be few. The nerves enter in large bundles at the apex, a few from the wall of the alveolus and a few over the alveolar border.

It will be seen then that this richly nourished membrane is very dense in structure, and arranged to literally suspend the tooth in its socket and hold it and the gum in position against all direction of the force of mastication.

Apical Pericementitis.

The term of apical pericementitis is applied to the inflammation of that portion of the peridental membrane situated about the root apex. There are two varieties of this affection, acute and chronic. An acute apical pericementitis is an inflammation just forming and characterized by all the pathological changes which occur in other acute inflammations.

There is a decided thickening of the membrane, and because of its bony surrounding it is a painful process.

When we remember that the sense of touch in the tooth lies in this membrane it can readily be seen why this is such a painful process.

Causes.

The causes of apical pericementitis most frequently lie within the pulp chamber, but not always. It is usually dependent upon the death and putrefaction of the pulp, but may be caused by a great variety of things such as a shock, severe use, unusual stress, irritating substances passing through the canal, following pulp extirpation, root fillings, too severe wedging and the use of the mallet in large gold fillings, taking cold, etc. I have occasionally observed that very rheumatic individuals sometimes suffer from disturbances of this nature, which is only temporary, rapidly passing away when the constitutional conditions are relieved. In its simplest form it is caused by the hyperemia or inflammation existing in the pulp tissue extending through the apex to that tissue.

In its severest form it is caused by infection from pulp putrefaction by pyogenic organisms.

Symptom.

The most prominent symptom and the one always present in acute cases is tenderness to percussion.

Patients complain of the tooth feeling long, occluding before the others, and for this reason they can locate it. There is usually some slight redness of the gum tissue opposite the apex and often slight tenderness

to digital pressure in the same region. There is an actual elongation of the tooth due to the thickening of this membrane forcing the tooth down from its socket. To percussion the tooth has a dull sound and if the pulp is dead there will be the absence of sensitiveness to thermal changes.

Treatment.

In acute cases the treatment is very simple.

Remove the cause and put the part to rest. If the cause lies within the pulp canal then that must receive first attention, and be thoroughly cleansed and, so far as the membrane itself is concerned, it will take care of itself.

Many teeth in this condition are lost or turned into chronic form by too frequent medication, especially with irritating agents; however, when the canal is filled with putrescent material it must receive careful attention in order to avoid running into acute alveolar abscess. This treatment has been fully described in the previous chapter and all I wish to say here is that careful instrumentation is the essential requirement in treating these cases together with mild non-irritating antiseptics, and rest. Do not be poking medicines in there every day for months; apply your remedy the first sitting, change it in 24 or 48 hours, and mechanically clean the canal, then reapply your dressing and allow to remain a week or ten days when, in the great majority of cases, the canal filling may be proceeded with.

Occasionally we meet with cases that do not yield so readily. Pain continues, and must be met by other means, such as counter-irritants with the pepper pads, tr. iodine, chloroform confined, or blood letting; sometimes opening into the apical space through the outer wall of the alveolus and lacerating the tissue will be helpful. This opening can be made by dipping a coarsely saturated plugger in 95 per cent carbolic acid and allowing this to touch the mucous membrane over the root apex; then rubbing off the white eschar, repeat until the bone is reached when, with a proper drill in the engine, you can penetrate the space painlessly.

This should be supplemented by the hot foot bath, 5 to 10 grains of quinine followed by small does of tr. aconite and tr. gelsemium, alternately, half hour for 6 or 8 hours, and at bedtime 10 to 15 grains of Dovers powder, followed in the early morning by a copious saline cathartic.

This same line of treatment is sometimes indicated in acute forming alveolar abscess.

In acute cases where there is no perceptible putrescence in the canal don't imagine you have an abscess at the apex because of the tenderness, and don't try to enlarge the foramen so you can force medicines through, but lay in a mild antiseptic and let it rest a week or two.

Where acute pericementitis develops while devitalization and pulp removal are in progress, it is probable that the operator is to blame. He may have been careless with his instruments as regarding their proper sterilization, or pushed one through the apex, or used irritating agents in too great a quantity in the canal. A case I had a few years ago illustrates the point I wish to make. A well known dentist devitalized a pulp in a central incisor in a gentleman of good health about the age of thirty-five. He removed the pulp and sealed in some oil of cloves and dismissed the case for a week. Patient returned in three days, tooth sore to the touch; dentist removed dressing, washed out and sealed in fresh dressing of the same. Conditions grew rapidly worse and he concluded he had some infection, so after changing again he sealed in oil of cassia because of its reputed great antiseptic power. This made matters worse and when patient returned he could hardly bear to have the tooth touched, but when the dentist did succeed in removing the dressing down came thin, watery fluid, and he at once concluded he had violent infection and left the root open. Up to this time eight weeks had been consumed, all of which time patient was in agony, but as he left the tooth open with no irritating oil in the canal it rapidly quieted down. Patient returned again and dentist sealed in his powerful antiseptic with like results, and patient removed the dressing himself, which the dentist had recommended him to do in case he had trouble. By and by there was real infection, as you can readily see from leaving this canal open. They battled along this way for a year and a half until the dentist became alarmed and feared serious necrosis and advised extraction, but it was a case where it would be the only tooth the gentleman had lost in his upper jaw and he disliked very much indeed to lose it. So he brought the patient to me for counsel, and after examination he turned him over to me to make a trial. I cleansed out the tooth thoroughly, dried it, sealed in a powerful non-irritating germicide, forcing a little beyond the apex into the cavity absorbed in the bone. And let me say that when I saw the case there was absorption beyond the end of the root almost as deep as the length of the root itself; a great pocket there; of course, it was filled with pus. The agent that I used in that particular case was a ten per cent solution of chinosol, which I will have occasion to refer to later. After carrying this medicine a little into the absorbed pocket beyond, I sealed the cavity and instructed the patient to return in five days, but if the soreness increased markedly, not to open it himself, but to return at once to me. At the end of five days the tooth was completely comfortable. I redressed it as before and dismissed him for two weeks. On examining the case this time I noticed the tissue rapidly filling in the absorption. I redressed it and dismissed him for two weeks more, at the end of which time I filled the root with no unpleasant

after effects. I have had occasion to watch the case and have seen it within the last two months and know that it has never given any trouble. This is typical of many, many cases.

There is often a little soreness following pulp extirpation and the same following pulp canal filling. Give it absolute rest, don't try to put a filling in or otherwise further irritate it. The point is, do not get any irritating oil or its vapor through the apex in cases where pulps have been devitalized and removed. If you should accidentally do so, just let it rest, do not disturb it, nature soon recovers.

Chronic Apical Pericementitis.

Acute apical pericementitis will usually naturally terminate in one or two conditions, namely, either in acute alveolar abscess or in chronic apical pericementitis, depending quite largely on the nature and severity of the irritant.

If it be mild nature, somewhat constant, not suppurative in its nature, it is very liable to terminate in the chronic form, to treat which often baffles the skill of the best practitioners.

Chronic apical pericementitis presents all the symptoms of the acute in modified form. Patients usually complain of much or little soreness, which may have extended over months of time continuously, or the tenderness may come and go every few days. Usually the gum is a little red over the apex and slightly tender to digital pressure. I have met several cases where there was a decided thickening of the bone over the apex, and many times there is a thickening of the cementum.

Cases.

The chronic form usually follows an acute attack where the irritant is mild and continuous, sometimes following pulp canal treatment, root fillings, broken broaches, and such, but most frequently is the result of very mild putrescence in the pulp canals where pulps die under fillings or from irritation of some kind, and very slowly disintegrates, just enough poison being formed to keep up a constant irritation.

Occasionally we run across a patient whose peridental membranes are very prone to inflammation. I usually find such an individual suffers from chronic inflammation of all the mucous membranes.

Treatment.

The management of these cases often try the patience of the operator. The technical procedure must be along the lines already mentioned. Relieve the tooth by grinding its occlusal surface or that of its opposite in such a way that the stress of closing the jaws will be borne by the other teeth. I sometimes get good results by sealing in the canal well up to the foramen a saturated solution of iodine in creosote, for its irritant

alterative effect. Its liability to cause discoloration is its chief objection, and so must be cautiously used.

Sometimes stirring up by passing a smooth broach through the foramen will be helpful. A 3 per cent solution of formaldehyde is sometimes helpful; anything that will cause a mild increase of the soreness will usually aid. These are the kind of cases that usually get sore soon after treatment is sealed and patients either open the approach or ask the dentist to. They should be encouraged to bear the added pain for a little time in the interest of permanent cure. What I said regarding treatment of acute cases applies here; do not keep changing the dressings every day, but leave one good dressing applied on cotton in the canal tightly sealed for two weeks at a time. Many times for a second treatment I have used a paste made of hydronaphthol with trikresol, and left it in the canals for six weeks, with gradual disappearance of symptoms and permanent cure as a result. No hard and fast rules can be laid down for treating these cases; the good judgment of the operator must determine how to manage each individual case that is presented.

Alveolar Abscess.

An abscess is a collection of pus within the tissues which is always preceded by a circumscribed destructive inflammation which results in the breaking down of the tissues in a given area. The term alveolar abscess has been arbitrarily restricted to those occurring at the apical portion of teeth or the apical portion of the peridental membrane, although strictly speaking any abscess occurring in the alveolus would be an alveolar abscess (Fig. 33). These abscesses are divided into two general classes, namely, acute and chronic.

Fig. 33.
Showing abscess on the side of buccal roots of an upper molar. (Barrett.)

Causes.

Acute alveolar abscess occurs when apical pericementitis becomes infected with pus forming germs. It is always dependent upon the death of the pulp. As all forms of apical pericementitis of any marked degree can only occur after the pulp is dead, so also we can only have alveolar abscess after the pulp is dead and infected. For here, as elsewhere, we can have no pus without pus producing micro-organisms. When the inflammatory condition around the apex becomes infected, usually inflammation rapidly increases, sometimes slowly, being governed here as elsewhere by the condition of the germs, of the local tissue and the general system.

This inflammation causes rapid swelling of the peridental membrane, causing the tooth to be lifted out of the socket, unless the condition of the bone beyond offers less resistance. As in severe cases of apical pericementitis the tooth is actually elongated; patients always complain of the tooth being long, being in the way, they strike it before they do the other teeth in occluding. And, as a matter of fact, the tooth is elongated, i. e., it is actually pushed down out of the socket by the swelling of the membrane at the apex. You can readily see why this must be such a painful process. Indeed, acute alveolar abscess is usually the most painful condition with which we have to deal.

We divide acute alveolar abscess into four classes according to the manner of the escape of pus. First, where the pus passes through the alveolus into the soft tissue, producing a rounded fluctuated tumor directly over the root of the affected tooth. This is the form most frequently met with and is most easily handled (Fig. 34). Second, where

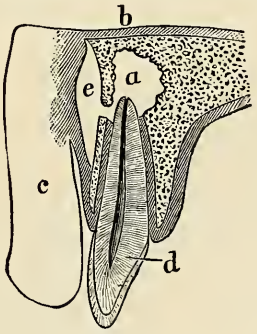

Fig. 34.
Acute alveolar abscess of upper central incisor, pointing on the gum. *a*, abscess cavity; *b*, floor of nostril; *c*, lip; *d*, tooth. (Black.)

the pus passing through the alveolus tears up the periosteum, a thing which often occurs (see Fig. 35, also Fig. 36).

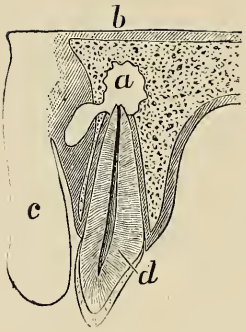

Fig. 35.

Acute alveolar abscess with pocket of pus between periosteum and bone *A*, abscess cavity; *b*, floor of nostril; *c*, lip; *d*, tooth; *e*, pus cavity beneath the periosteum. (Black.)

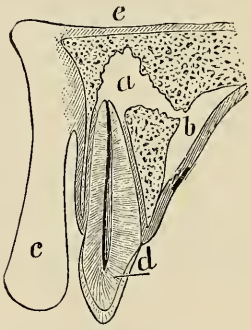

Fig. 36.

Acute alveolar abscess with pus pocket between periosteum and bone of palate. *A*, abscess cavity; *b*, pus cavity beneath periosteum; *c*, lip; *d*, tooth; *e*, floor of nostril. (Black.)

The pus here has passed through the bony process and has taken up the periosteum of the bone, which can usually be determined by finding a broad, long, flat tumor, sometimes extending two or three teeth mesially and as far distally as the last tooth. When you cut in with a lancet you discover an area of some size denuded of the periosteum.

I have seen many cases hold pus in such a sac for days and show a tendency to pass along towards the gingiva and discharge. These cases are not so frequent as the others, but every practitioner meets a considerable number every year. Usually when pus is discharged they get well readily; the periosteum reattaches itself to the bone. After the pus has discharged it will usually lie back upon the bone and reattach itself.

Usually, I say, but sometimes these cases terminate seriously by the destruction of large areas of bone as a result of tearing away the periosteum.

The third form is where the discharge occurs along the side of the root between the peridental membrane and the tooth. This is by far the least frequent of all forms of alveolar abscess, I am glad to say; but it is

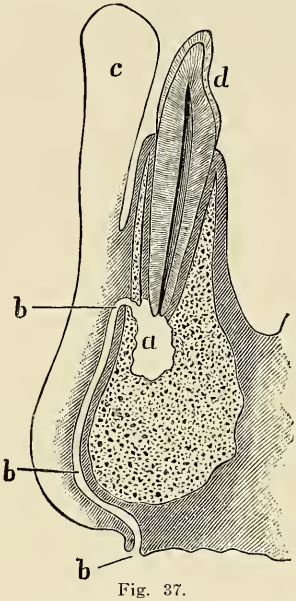

Fig. 37.

Chronic alveolar abscess at root of lower incisor, discharging under chin. *A*, abscess cavity; *b*, *b*, *b*, fistula; *c*, lip; *d*, tooth. (Black.)

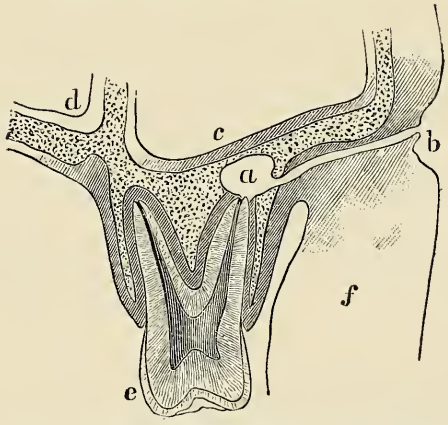

Fig. 38.

Abscess of buccal roots of upper molar discharging on the face. *A*, abscess cavity; *b*, point of discharge on face; *c*, antrum; *f*, lip; *e*, tooth. (Black.)

also much the most difficult to handle and when chronic is liable to be mistaken for pyorrhea alveolaris. Fourth class is where the pus, instead of making an exit in any of the ways indicated, follows along the sheath of some muscle, or its fibres, until it finds an easy exit. In some cases it may follow until it reaches the muscle attachment to the bone and then its exit at that point. You may set down a rule that pus will always go in the direction of least resistance. Very often it comes to the skin in all sorts of places, particularly around the lower jaw (Fig. 37), upon the cheek (Fig. 38), under the eye, in front of the ear; when they open on the face they make ugly scars.

Symptoms and Pathology.

Tooth feels longer and strikes occluding teeth before others do. At first the striking may give relief, i. e., when the inflammation is first started up, to bite on the tooth may feel good. But it soon becomes extremely painful; the slightest jar causes pain. In these acute cases the patient can scarcely lie down; temperature and pulse go up; agony increases every minute until it becomes almost intolerable. Let me say that in the great majority of cases of acute alveolar abscess you can tell what the patients are suffering from by seeing them come into the office. You find the patients coming in walking on their toes; they don't want to set their heels down because the slightest jar causes pain. And, of course, they usually have the expression of agony written upon their faces. As the inflammation increases the bone around the apex begins to absorb to accommodate the swelling membrane and the forming pus. On and on it goes, pus forming a little faster than space to accommodate it, and the temperature continues to rise. By and by one or more Haversian canals running into this space begin to enlarge or absorb, the pus follows in these canals until by and by it reaches through the bone to the soft tissue. It usually goes towards the labial, rarely towards the lingual, although occasionally. When the soft tissue is reached the swelling begins. Pus goes in the way of least resistance. The swelling continues sometimes to enormous size and shape. Pus continues to burrow in the way of least resistance until it finally approaches the surface of the gum, which usually puffs out in the form of a rounded tumor which points more and more until finally it breaks through and pus is discharged and pain subsides. Indeed, pain begins to quiet down as soon as the pus passes through the bone and the swelling begins.

Treatment.

In all acute cases the first thing to attend to is the forming tumor. Where there is a well rounded tumor forming on the gum over the root apex the only thing needed so far as the tumor itself is concerned is to

let the pus out by cutting with a lancet at the most dependent part. I wish to emphasize the idea of cutting by drawing the edge of the blade along the bottom of the tumor rather than attempting to puncture it with the point of the lancet. The latter is usually painful because of the pressure exerted on already distended and tightly drawn tissue.

After this has been done the case should be allowed to rest for a few days until tenderness has subsided, when the offending pulp chamber can be opened and proper treatment proceeded with. In the second class it is necessary to make a free long incision at the most dependent part of the tumor, press out the pus and thoroughly irrigate, examining to see if any necrosed bone be present. The offending tooth then is allowed to rest until soreness has subsided. I have had a number of cases where these alveolar abscesses had literally torn away the periosteum from the alveolus around two or three or maybe four teeth, and the whole mass of bone dead, so that the teeth and bone and all would come out with the slightest effort. This is a thing that is likely to occur in cases of serious accident, people meeting with traumatic injuries, etc. These cases where surgical interference is sometimes necessary, but frequently, and, indeed, usually, the bone will be thrown off in a large mass, and if the periosteum remains alive it will gradually rebuild the bone. This is a most remarkable thing, and I have seen some of the most remarkable recoveries where the bone became necrosed and sloughed off a large area. That is the process by which all fractures are healed, so that you do not need to worry in those cases where the periosteum is alive; but if the periosteum is gone, then your case is hopeless. In these cases the parts must be made clean, surgically clean, all dead bone removed, and kept clean until recovery is complete. In the cases that I have just illustrated in Fig. 35, when you make your opening with the lancet and have all the pus out, always take pains to wash out such a case as this, cleanse it out with carbolized water, differing in that respect from the rounded tumors. The irregular places in which pus may have caught, as it were, make it necessary that you wash it out with carbolized water. Always do that after opening this kind of a case. In the third variety there is usually no tumor, although sometimes the pus makes its way out into the gum tissue near the gingivus, in which case the tumor should be opened and drained. Where the discharge is along the peridental membrane, making its exit between the gum and the neck of the tooth, the pulp canals must receive immediate attention.

In the fourth class, where pus is attempting to make an exit on the skin surface, especially about the face, very careful attention, too, is required. When a case comes to you with the tissue much swollen and hard in certain areas, never poultice—please bear that in mind—but dis-

courage its coming to the surface by free incision in the mouth. Let us illustrate: Take a case where the swelling is on the lower jaw, perhaps involving the submaxillary gland. The tissues are very much distended and a certain area, perhaps the size of a twenty-five cent piece, is very hard and has already begun to get red or purple in that particular region. If you poultice that a few days it will break on the outside. Instead of that, as soon as you see the case you take a lancet and make a free incision into that abscess from within the mouth, drawing out the cheek and making an incision clear along the periosteum of the bone until you reach the abscess and get the discharge within the mouth. Oftentimes cases will come to you too late for that sort of thing, that have perhaps already broken and are discharging on the face. Then, in that case, the first thing to do is to cut off that discharge as soon as possible, either by the extraction of the tooth, in case the tooth can be spared, or making this incision in the soft tissue from within the oral cavity, thereby cutting off the discharge from the external surface.

The case may come to you before the pus has appeared beneath the surface of the gum; in this acute condition when the tooth is so sore you can scarcely touch it, and a collection of pus in the apical space; it hasn't even penetrated the bone yet; or it may have gone far enough for the pus to have penetrated the bone and no farther; indeed, that is quite a usual condition that we meet with. In this case, when you feel certain that the pus has already passed the bone, inject a little cocaine and make a deep incision over the root clear to the bone until you find the pus. You usually will be able to detect whether the pus has escaped through the alveolus by the finger gently pressing upon the soft tissue; you will get the slight fluctuation that always presents after the pus has passed beyond the bone. In case the pus has not reached the periosteum, is still confined within the bone, what shall we do to relieve that case? That case is more difficult to handle. Many dentists send patients away to poultice and await swelling and breaking on the surface, which seems a cruel thing to do. In this class of cases it has been my custom to open into the apical space, either by the use of a strong knife passed through the soft tissue and bone, after having anesthetized the parts with cocaine, or, in case that seems impracticable, the bone is very heavy and I do not succeed in that method, or for any other reason I decide that this is not practicable, then the method that I use is the same as described for opening the apical space in apical pericementitis.

The advantages of using this carbolic acid and plugger method are two. It prevents hemorrhage and can be done painlessly. Care must be taken not to drill into the tooth root.

When it is possible to open into these cases directly through the tooth

or cavity at the first sitting, it is always advisable to do so. In case there is no cavity, we open upon the lingual or occlusal surface. But often the teeth are so sore as to make the operation impossible, especially if we have to open in through sound tooth structure or a filling. Of course, you readily understand that if there was a large cavity and the pulpal wall was thin, there would be very little difficulty, even though the tooth was sore, in making that slight opening; but in case it is necessary to drill through sound tooth structure like this, or a filling, they are often so sore as to make it quite impossible.

I want to suggest a few methods that will often make this possible. After you have everything in readiness for opening, place the finger or thumb of the left hand on the crown of the tooth and begin by pressing gently, increasing gradually until you have accustomed the tooth to it; then hold it rigid and open directly with a rapidly revolving drill. If it were a molar tooth on the lower jaw I probably would hold it with my thumb. I sometimes take the sore tooth and its neighbor between the thumb and forefinger in a tight grasp, thereby holding firm and preventing much of the jar of the drill. Another method is to surround the sore tooth and its neighbors with plaster of paris. That is a method I use especially where teeth are very loose, and it is very serviceable.

Let us take, for example, a lower first molar, all neighboring teeth in position. We decide that it must be opened and we are going to try to open it rather than take the chances of opening through the soft and hard tissues. First dry surfaces; pack toward the tongue and toward the cheek with cotton to keep the secretions away; then mix plaster with a little salt in it so it will set quickly, and cover the second molar and wisdom tooth, if they be present, clear down to the gum on either side as far down as you can; also cover the sore tooth and the two teeth anterior to it; then before it gets real hard remove the plaster from the occlusal surface of the tooth upon which you are going to drill; then let the whole thing set very firmly. This will often help when the tooth is quite sore. Try it and see. Your success will depend upon the perfection with which you get your plaster around the tooth, so it is in contact with all the tooth and the gum, thereby distributing the pressure from one tooth to five teeth. That I suppose you would call making a temporary splint. Where teeth are affected with pyorrhea and are very loose and it is necessary to open into the canal (many of these come to us with pulps dead), that is the method that I use almost universally. Then another method is to ligate the teeth, beginning with the second molar, tying your ligature on the mesial surface of it, then around the sore tooth and ligating again on the mesial surface of that, and then around the second bicuspid in the same manner; passing your ligature back and forth in that way bind and

lock the teeth solidly together. The objection to that method is that it is sometimes painful in tying the ligature. Otherwise, it is very useful. Another method that I use very frequently is to tie the ligature strongly around the neck of the tooth to be drilled into, and then with the thumb or the forefinger I catch a loop of the ligature and gently lift up on the tooth, increasing until I get a good strong pressure, literally lifting the tooth away from the swollen and irritated peridental membrane; and drill as before. Another method that I use frequently is to take the ligature and tie it around the "sick" tooth, making the knot distally; take another one and bringing it around the tooth and making the knot mesially; that gives two ligatures, one to the mesial and one to the distal. Then take ligature and pass it from the mesial around to the distal of the second molar, and tie it. Then I do the same thing with the second bicuspid, having the ligature tied between the two bicuspids, bring ligature that is on the distal surface of the second molar up over the occlusal surface and the one on the distal surface of the first molar and tie over the occlusal of the second molar and likewise, the one on the mesial of the second bicuspid and on the mesial of first molar tied on the occlusal of second bicuspid. You see, this actually lifts the sore tooth out of the socket, a thing that is very simple to do, is not painful, and is very successful.

There are many cases presented in which we fail to give relief either because of the nervousness of the patient or the severely painful condition present, and all we can do is to administer such general remedies as will aid the sufferer in accordance with the plan suggested in the treatment of painful pericementitis. One thing in addition can be done, and that is to make hot water applications to the face, which will tend to relieve the pain and at the same time hasten the pointing of the abscess. We should keep in mind the danger of hard swollen areas breaking on the face, and discontinue such applications where this tendency is shown, although my experience has been that where moist heat is used that danger is not so great as where dry heat is used. In all cases we should seek the first opportunity to make an opening either through the gum or into the pulp chamber, and thus afford relief, and avoid those serious conditions which are liable to happen.

Chronic Alveolar Abscess.

The tendency of all acute abscesses is to become chronic; when they discharge their pus upon the surface, either of the skin or the gum, they frequently heal over; then in a few days they break and discharge again, and repeat this process every few days for weeks and years. Usually patients with such cases think nothing of it at all; usually painless. After they have discharged the first time the patient will come to you, perhaps

telling you that a little swelling occurs in a certain region of the mouth; it puffs up, they pass their fingers over it, it breaks and that is the end of it, until it recurs.

It is in this class of chronic cases that we have the worst burrowing of pus. Little by little the pus will burrow along the periosteum of the bone. Take a case where it has discharged upon the gum and heals over (Fig. 39). The next time that Nature makes an effort to force the pus

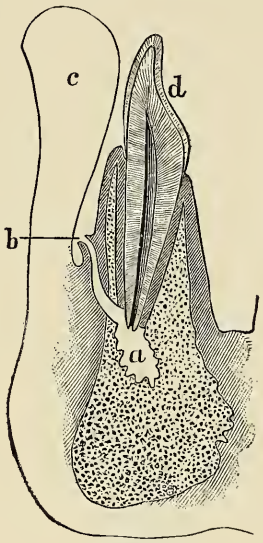

Fig. 39.
Chronic alveolar abscess at root of lower incisor. *A*, abscess cavity; *b*, fistula discharging on gum; *c*, lip; *d*, tooth. (Black.)

out through that tract it will drop down a little lower each time, until by and by you will have pus burrowing in all sorts of directions (See Figs. 37 and 38). I have had some very remarkable cases. I remember one case which was the most interesting of all. It was during the early years of my practice. I was called upon to extract a lower wisdom tooth on the right side. The tooth was badly broken down, was not paining at all, but the odor from it was offensive, and I found that the wisest thing to do was to extract it. The patient told me that she suffered at times with what appeared to be a sort of paralysis of the right arm, i. e., it would be all numb, and the same on that side of the neck. I did not associate it with the tooth, but after extracting the tooth and washing out the socket I found that I readily forced water out just at the clavicle, and upon inquiry I found that she had a place that was discharging there.

She thought it was a tubercular gland and had it opened twice by a surgeon and the bone scraped. I flooded it through with carbolized water and then washed it through with a twenty-five per cent emulsion of carbolic acid, when it healed up without trouble.

I remember another case that I had where the discharge was just under the lower jaw about the region of the submaxillary gland. I was doing some dental work for this case also; patient did not come to me for treatment of this, believing that it was a cancer there. She had it opened and scraped any number of times; it would heal up and be all right for two or three weeks and then it would puff out again and break and discharge. On making a filling in a lower first molar I discovered that the pulp was dead. I opened into that tooth, cleaned out the root canal and had no difficulty in washing through the root of that tooth right through this opening. In that case the pus had gone directly through the bone; the channel was directly through the bone, appearing on the under side. In that particular case we were able to save the tooth and heal up the difficulty in the jaw.

But oftentimes in these cases where pus has burrowed either through the bone or along the periosteum for some way before it discharges we

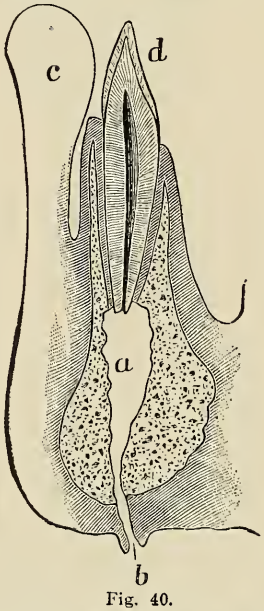

Fig. 40.

Chronic alveolar abscess, lower incisor with abscess cavity extending through the body of bone, discharging on chin. *A*, abscess cavity; *b*, mouth of fistula; *d*, tooth; *c*, lip. (Black.)

have serious necrosis of the bone, and it does require something of a surgical nature. I think perhaps you have all read of the interesting case of Dr. Black where the pus had burrowed from a lower central incisor directly through the jaw (see Fig. 40). He was consulted in the matter by the surgeon who was handling the case and asked if he thought the tooth might have anything to do with it, and he said he would be decidedly of the opinion that it might have. Then they wanted to know what they would look for to tell; he told them that perhaps the tooth was loose; perhaps discolored or something of that kind; perhaps it had a history of being sore once upon a time, or something of that kind. They made an examination and decided that the tooth was responsible and extracted it and washed down through the socket underneath the chin. Then they made an arrangement whereby they tied a string on a sponge, and saturated this sponge with medicine and pulled it through, and then pulled it back again. They told the patient it was a very serious case and had her coming every day to run this sponge through to keep it clean and open. It happened that she lived quite a little ways from the office, and there came up a very severe storm, making it impossible for her to come out, and I believe she didn't come for three days, and when she did come it was all healed up, which is the usual thing. These cases get well readily usually where the pus has burrowed without much difficulty, especially where it has not met with much resistance.

It is probable that these cases discharge more frequently in the antrum than we imagine (Fig. 41). If you will examine all the skulls

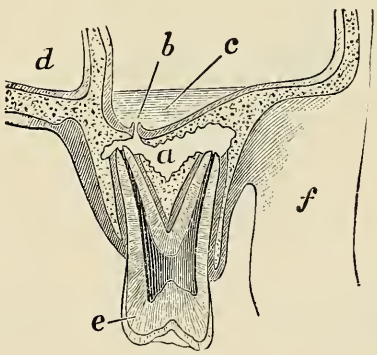

Fig. 41.

Alveolar abscess at the roots of upper molar discharging into the antrum. *A*, abscess cavity; *b*, mouth of fistula; *c*, pus in antrum; *d*, nostril; *e*, tooth; *f*, lip. (Black.)

you get an opportunity to, you will be surprised at how thin the floor of the antrum is in many cases, and I am sure often we have these cases penetrating the antrum when we do not realize it; because of the absence

of pain patients frequently do not know of the trouble until severe empyemia results. In chronic cases where pus is discharging on the gum or other surface careful exploration should be made through the sinus with the silver probe to locate its direction and discover if anything unusual is present. I wish to emphasize the value of the silver probe as a diagnostic aid in these cases where the point of discharge is upon the face or neck; in addition to cutting off the sinus and emptying it in the mouth, some attention must be given to the scar. It should be dissected away from the periosteum, to which it is usually attached, packed, and if need be held out in proper position with a long silver pin passing through the scar tissue, resting on the surrounding parts in such a way as to hold the depressed part out level with the adjacent tissues. The packing must be kept up until healing results from that side. In all cases of alveolar abscesses, the offending teeth—the teeth whose pulp have died—must receive proper treatment and the canals thoroughly filled if a permanent cure results.

A word should be said here regarding molar teeth. It is sometimes seen that only one root canal is discharging on the gum, or perhaps the two buccal roots in an upper molar, while the lingual root may be discharging in the palate or only presenting a tumor-like swelling (Fig. 42), which swelling often resembles aneurysms. There is danger of mistaking the latter for a forming abscess (Fig. 43).

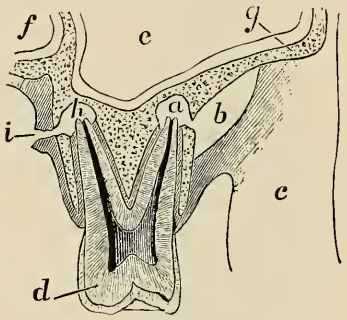

Fig. 42.

Acute abscess at buccal roots and chronic abscess at lingual root. *A*, acute abscess cavity; *b*, pus cavity between bone and periosteum; *c*, lip; *d*, tooth; *e*, antrum; *f*, nostril; *g*, molar process; *h*, chronic abscess discharging at *i*. (Black.)

Aneurysm.

"Aneurysm does not often come under the observation of the dentist, but it does occur occasionally in the tract of the posterior palatine artery, and in this locality I have known it to be mistaken for an alveolar abscess with serious results; therefore never incise tumors on the hard palate

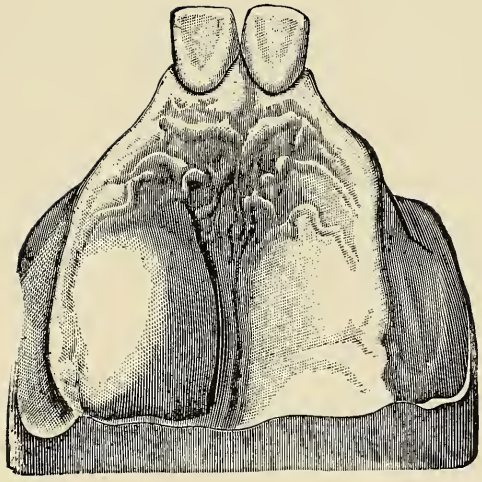

Fig. 43.
Aneurysm of posterior palatine artery. (Marshall.)

without first having given them a careful examination to determine certainly their nature.

In order to differentiate between an alveolar abscess pointing on the hard palate and an aneurysm of the palatine artery, make pressure on the proximal side of the artery from the tumor, and if the tumor decreases in size the indications point to an aneurysm.

Again, place the thumb on one side of the tumor and the index finger on the other; if there is decided pulsation, which not only raises the thumb and finger, but at the same time perceptibly separates the two, this also is an indication of an aneurysm. Simple pulsations do not certainly indicate an aneurysm. As a tumor overlying an artery may give sufficient impulse to the tumor, so that the result of the heart's action may be felt through its substance, if the tumor be not too massive and the artery sufficiently large.

To come to a positive diagnosis, it may be necessary to aspirate a part of the contents of the tumor. This may be done by inserting the needle of a hypodermic syringe into the tumor and then withdrawing the piston. If it is an aneurysm the barrel of the syringe will contain arterial blood; if an abscess it will contain pus. This precaution may prevent a serious result, and is advised in all cases where there is doubt." (Gilmer).

Blind Abscess.

The term blind abscess is applied to those cases where there is a pus cavity about the root apex with no fistulous or other opening to the external surfaces. They are for the most part chronic cases that lay dor-

mant, although they may occasionally have periods of tenderness. They seem to be very slowly progressing, absorbing the bone little by little, and show no tendency to heal.

The pus is usually of the laudable variety, although very liable to take on the septic thin serous or watery form, in which case acute conditions are set up, with more rapid destruction of both bone and soft tissue. This is the thing that is so liable to happen when the pulp chamber of a tooth in this condition is opened for the first treatment, and requires the same care as is necessary in putrescent cases, which has already been alluded to.

Treatment of Pulpless Teeth.

For convenience of treatment description it seems advisable to divide all these varieties of alveolar abscess into four classes.

First—Those cases where the pulp canals are open and have been exposed to the fluids of the mouth.

Second—Cases where pus is discharging through a fistulous opening on the gum.

Third—Those cases where the discharge of pus is at some point distant from the root of the offending tooth.

Fourth—Blind abscesses and other dormant cases.

Each class requires some important modification in detail of treatment. In the first class are those cases where the pulp has died as a result of exposure by caries and gone through the stages of putrescence into acute alveolar abscess and perhaps on to the chronic form.

The management of these cases is among the most difficult, for like many dormant cases they are very prone to take on active condition when treatment begins.

The first step is to remove all the decay from the carious cavity and pulp chamber, opening up thoroughly to get free access. This can usually be accomplished best with spoon excavators and inverted cone bur after the enamel has been chiseled to outline. This part can usually be done without the application of the rubber dam, which will allow free flushing with warm antiseptic solution, washing out all the débris as fast as it is loosened in excavating, keeping in mind always that it is important that nothing whatever shall be forced through the apical foramen. The next step is to apply the rubber dam. Then flood the cavity and chamber with alcohol, absorbing with cotton and reapplying until all is clean and dry; flood again with alcohol, this time carefully working it down into the canal with a smooth broach and then wash out all débris; the next step is to dry first with alcohol on cotton, then dry cotton and finally with warm air.

In this dry warm condition a non-irritating antiseptic should be introduced loosely on cotton, for which purpose oil of cloves, 1-2-3, trik-resol, oil cloves and eucalyptol. Creosote has also proven a good agent, and sealed with gutta-percha in such a way as not to allow anything forced through the foramen. If all keeps well this agent should be allowed to remain for four days.

At the second sitting the dam is applied, the field of operation cleansed, the canals reopened, and this time thoroughly cleansed with proper broaches, so that all walls of the canals are free from any clinging débris; alcohol is again used to float out all loose material and dry the canals under the same precaution as before. A second sealing of the same kind of agent allowed to remain 10 days will usually effect a cure and prepare the tooth for root canal filling. In the management of these cases if acute conditions should set in during the progress of treatment the patient should return immediately, the case be reopened and treated as an acute apical pericementitis.

The second class of cases includes both the acute and chronic forms. I have previously stated in this chapter that the first thing to do in acute cases is to get an outlet for the forming pus and dismiss the case until tenderness has subsided. When the patient returns for the second sitting and the opening, if made on the gum, has healed, you proceed exactly as described in Class 1; if said opening still remains the procedure is the same as for the ordinary chronic case with discharge on the gum. The first step is to clean the carious cavity if one is present, and adjacent teeth, then apply the dam and thoroughly cleanse and disinfect the field of operation, particularly the tooth to be opened. The second step is to secure convenient access to the chamber and canals, floating out all débris with a solution of bicarbonate of soda in distilled water; the canals should then be mechanically cleaned and again washed with the above solution, taking plenty of time to wash out all débris possible from the tubuli. The next step is to thoroughly dry the canals and disinfect and deodorize the dentine with free application of oil of cloves, using gentle heat to force it throughout the dentine as far as possible; the object is to cleanse and deodorize the dentine before any agent is used that will seal the tubuli filled with filth. The excess of oil is removed by alcohol and the tooth made ready for washing through the fistula. A smooth broach is used to make free the opening through the apex. If the approach to the canals is not so shaped as to prevent the escape of solutions under pressure it should be so arranged either by the use of gutta-percha or cement, as seems best.

The next step is to force a saturated solution of bicarbonate of soda through the canals and out the fistulous tract freely; for this the Dunn,

or Farrar syringe or hypodermic syringe with platinum point are well adapted; over the needle is placed a tapering rubber cone, the needle carried into the canal and the cone held with a strong pair of cotton pliers against the prepared orifice. Force enough is exerted on the plunger to thoroughly wash the abscess contents out through the fistula. It is important to empty the abscess of its pus before using coagulating agents.

The next step is to dry the canal and fill it with cotton soaked in 1-2-3 or 95 per cent carbolic acid, or phenol-sulphonic acid, depending on the conditions about the apex. Some cotton smeared with vaseline should be placed around the opening on the gum to prevent severe burning in case an excess of the escharotic escapes. The medicine in the canal is then forced through, using a piece of soft rubber and an amalgam plugger as a plunger. When the agent appears at the opening on the gum it can readily be detected by the white appearance of the orifice of the fistula.

At this point it has been my practice, in bad cases, to hold a soft piece of rubber over this opening and again pump, thereby forcing the agent into every corner of the abscess cavity. A mild dressing is sealed in the canal for a week, when if all goes well the root may be filled. In some cases one repetition of this treatment through the fistula may be wise. This procedure will bring the desired result in the great majority of cases, but for reasons which I shall presently point out some cases do not get well.

Occasionally one of these cases is met with where it is not possible to force anything through the apical foramen. When possible this should be opened, but this is an operation that requires skill. A Downey or a triangular piano wire broach are the best instruments to use for this purpose, and even with these there is some danger of breaking in the foramen. The operator should work with the idea of such a possibility in mind and proceed very slowly, frequently withdrawing the broach to make sure that it should not bind and break. As I have said, it is desirable to make such an opening when possible, but there are some cases where for various reasons this cannot be done. This treatment, then, must be the same as in Class 1, and in addition carbolic acid, 1-2-3, or phenol-sulphonic acid are carried into the abscess through the fistulous opening with the syringe. In many cases this will be sufficient (see apical cases). In the third class of cases, where the discharge is at some distant point, the treatment procedure is exactly the same as for Class 2, with this exception, namely, escharotics must not be forced through long distance of fistulous channels. It is safer practice to cut into the channel as near the tooth as possible and treat the tooth as though the discharge was at this point. The treatment of the rest of the channel and the external opening when on the skin has already been outlined in this chapter.

The fourth class—blind abscesses and dormant cases—often try

men's souls, because of the great liability of stirring up a hornet's nest. For the most part they are teeth whose pulp have died under filling or from traumatic injuries. I read a paper before the Chicago Dental Society in 1889 on this subject which was published in the *Dental Review* for May, 1900, from which I quote.

Every practitioner constantly meets with teeth whose vitality has been lost from causes that we do not understand.

That teeth sometimes lose their vitality and give no disturbance for years is a fact well known to all. Why this is so cannot always be accounted for. Sometimes the reasons can easily be understood. When a pulp dies it remains comparatively harmless until it becomes infected by micro-organisms—for without them we will have no putrefaction. If a tooth has no external opening into the pulp canal, then micro-organisms must enter—if they enter at all—through the apical end, and their only way of getting there is through the medium of the circulation. The blood does not normally contain micro-organisms, therefore it is only by accidents such as functional disturbances of nutritive processes, that they are enabled to live therein. When they gain entrance into the system in sufficient quantities and conditions are favorable they seem to immediately seek out dead or dying matter in which to grow and multiply. This explains why many teeth containing dead pulps, though remaining quiescent for years, suddenly develop acute conditions ending in acute and finally in chronic alveolar abscess.

When micro-organisms enter a pulp canal which is filled with dead matter we do not always have appreciable physical disturbances. In many cases we find foul smelling root canals when there has been no disturbance, even though the canals were closed. The understood reasons for this are three:

First—The animal cells surrounding the part affected may be sufficiently active to literally digest the micro-organisms and hold in check their putrefactive process.

Second—The system may be able to readily carry off the products of putrefaction.

Third—There may be present in the blood certain antitoxines which reduce the activity of the putrefactive process or neutralize its effects.

These reasons explain why teeth containing imperfect root filling remain quiet for years and other cases give occasional slight disturbances, which pass off and remain quiet for another period; then again the trouble is repeated and again ceases, and so on for years and years.

Why is it, when we open into the pulp chamber of teeth containing dead putrefactive matter, that we so often start up violent acute conditions? Why is it that we sometimes stir up a veritable "hornets' nest"?

I can only answer in part: We disturb the balance of power existing between the putrefactive process on one side, and the animal cell, antitoxines, or systemic conditions, on the other. We do this in four ways: By introducing fresh infectious material, or by forcing septic material through into the apical space, or by introducing some agent that will interfere with the local animal cell activity or admitting air.

There still remains unexplained many disturbances that follow the opening of certain cases. There is room for further study on these cases.

In certain cases after the death of the pulp, the apical foramen becomes closed either by deposit of cementum or by the peridental membrane growing tightly over it. All that is needed in treatment of these cases is to remove all dry material, moisten walls with a mild disinfectant, dry and fill immediately.

In a large number of cases examined every case containing moisture contained also pyogenic germs.

In those cases where there is no tenderness of tooth or tissue adjacent to the apex of the root, and no opening into the pulp chamber, after adjusting the dam, and disinfecting the tooth externally and surrounding tissues, I drill into the tooth almost, but not quite to the pulp chamber, and seal therein a mild penetrating antiseptic and let remain forty-eight hours. I now use my trikresol, oil of cloves and eucalyptol mixture—equal parts.

At the next sitting I enter the pulp chamber at its horn, using a small sterilized bur, cutting in such a way as not to produce the slightest pressure upon the contents of the canal; then I lay a piece of cotton containing some of the above solution over the opening partly to disinfect and partly to absorb the liquid present; then I carefully enlarge the opening and draw off the contents of the canal, which can be done nicely by the careful use of the hypodermic syringe, and dry thoroughly; next I lay a potent diffusible germicide and seal; care should be taken not to produce the slightest pressure. I allow this to remain thirty-six hours, when the case is again opened, now thoroughly—canal mechanically cleaned, reamed out and dried thoroughly. A non-irritating disinfectant is then introduced clear to the apex of the root, and volatilized by heat, then more introduced, sealed and allowed to remain a week or two longer. At the next sitting if canals are sweet and dry, and there has been no conscious disturbance, the roots are dried and filled.

In cases where there is tenderness to pressure, I open immediately, as before described, remove as much moisture as possible, and lay into the pulp chamber a potent germicide, seal with cement and drill a small "vent hole" through the cement and dismiss the case for twenty-four hours, after which I cleanse and dry the canal, as before described. If

tenderness continues I use creosote and iodine for second treatment, carried well up to the apex, seal, and allow to remain a week. In anterior teeth care must be taken not to introduce so much of this agent as to discolor the teeth. I wish to recommend this agent for cases of abscess in syphilitic patients. If tenderness now has disappeared I dry and fill immediately. If tenderness continues, however, as sometimes occurs, I treat with 12 per cent sulphuric acid, forcing carefully, a little through the apex, allow to remain twenty-four hours, and follow with mild antiseptic and dismiss for two weeks, when, unless a large absorption exists around the apex, the root may be filled. The sulphuric acid is recommended for the double purpose of dissolving any slight uneven, sharp points of foreign deposits on the root and destroying the so-called pyogenic membrane existing about the apex.

Since following the above plan I have had very little trouble, but when acute disturbances do arise the case must be treated the same as indicated for Class 1.

Special Cases.

After all I have said there yet remains many cases belonging to none of these classes and some in each of the four classes that do not yield to such comparatively simple measures, and sometimes cause a great deal of anxiety. For convenience of treatment description I place them in three classes.

First—Those cases where there is considerable absorption of bone around the root apex.

Second—Those cases where there is some absorption of the root end.

Third—Those cases where there is serumal calculus deposits on the apical end of the root (Fig. 44).

Fig. 44.
Serumal calculus covering the apical third of root of bicuspid.

In all of these cases there is more or less absorption of bone around the root apex, which can readily be detected by exploring through the sinus or by the amount of discharge coming through the root canal upon opening it up.

The nature of the absorption can, to some extent, be determined by the nature of the discharge. If it is thin, watery, yellowish, with little granules of bone mixed in, you can be pretty certain that caries of bone exists. If thick, rich pus, simple absorption. If this yellow is streaked with blood, no granules, you can count on a roughened root end, which should a little later be confirmed by exploring through the external opening.

I always proceed in the management of these cases exactly as I would for any chronic abscess, namely, open up canals, drain, dry, sterilize, deodorize the dentine; then if there be a fistulous opening I wash through with the bicarbonate of soda water, followed by 95 per cent of carbolic or 50 per cent phenol-sulphonic acid, using the latter if I am certain of considerable bone absorption. If it has been a bad pus case I next seal in 10 per cent chinosol for a week; if it is not such a case, but instead thin ichorous fluid present, I use paraform, or creosote and iodine well up to the apex. If there is no fistulous opening then I use the same treatment, except, of course, I do not wash through, but instead I force a little 50 per cent phenol-sulphonic through the apex into the space beyond. When the patient returns I always closely observe the conditions. Is there any further discharge through the fistula or down into the canal? What is its nature? Is the pus controlled? Does hemorrhage occur down into the canal? What does the blood look like? Is it rich red, showing that new granulations are present and the healing process nicely begun?

If I am favorably impressed with the progress of the case then I seal in a mixture of trikresol, oil of cloves and hydronaphthol and leave from two to four weeks, when I expect to fill the root canal. If there be a little thin yellow serous fluid weeping down into the canal on the second visit, and especially if there is a little soreness of tooth, I seal in creosote and iodine carried well up toward apex, absorbing excess to prevent discoloration. I will leave this from two to four weeks, when I will expect to fill the root canal. Occasionally I treat one of these cases a third time, but rarely. At this point I want to say that as a rule we over treat teeth, treat them to destruction. I know men who have these cases running to them every other day for weeks and months. Do you know such treatment does more harm than good? Be thorough in the detail of your work and use proper remedies and give nature time in which to make the recovery. If the cases do not progress favorably I fill the root canal and proceed to treat from the outside, for the reason that I probably have one of the

three conditions already described. For the last three or four years I have been studying these cases carefully and examining teeth removed by our extracting specialists, and in 85 per cent of the teeth removed, because chronic abscesses could not be cured, I found the trouble was either root roughened by absorption or deposits of calculus.

It has been my observation that most regular practitioners are either afraid or do not know how to make these explorations. The method I follow is this: First, I carry my index finger along the gum over the root apices both lingually and labially, using sufficient pressure to detect any tender, soft, or springy spots. If the case be long standing and considerable amount of absorption, I will usually find a spot over the apex where the bone has disappeared or has been so absorbed that only a thin plate remains, which will readily spring in, showing the absorption underneath. Next I inject a little cocain solution into the gum over the apex, and with a good strong lancet cut into it, making a good, generous opening, which with a little experience can be done painlessly; then with proper shaped instruments I explore every nook of that pocket, as well as the end of the root. If I am in doubt as to the conditions present, I will pack with antiseptic gauze and leave twenty-four hours, when if a little care is observed in removing the gauze I can readily see into the pocket and know for certain what has taken place and proceed accordingly. In cases like Class 1, the treatment is very simple. If it fails to heal readily from treatment as described through the root canal, which frequently occurs, I fill root canal and then make the generous opening as before mentioned; next pack the pocket with cotton saturated in cocain solution, being careful to so place loose cotton around the opening as to absorb any cocain that may exude.

I leave the cotton pack in the pocket for about five minutes, when I proceed to thoroughly curette the pocket, scraping all rough or dead bone (rather take a little more than necessary than not enough). While scraping I flood with cassia water, keeping it clean, so I can see exactly what I am doing. If the case should belong to the second or third class I proceed in exactly the same manner, and in addition I scrape off the serumal calculus and smooth up the root apex. In the second class of cases I resect the root end, cutting away all that portion that is roughened by absorption and at the same time make the end of the root round and smooth (see Fig. 69). This completes the surgical part of the work. There only remains the after treatment, which is very simple, and consists packing with plain gauze saturated with 25 per cent phenol-sulphonic acid, leave for 48 hours, remove pack, wash with cassia water and pack with antiseptic gauze, preferably aristol; repeat every third day for a couple of weeks, when the case should be well. The important point in

the treatment is to keep antiseptic and compel healing from bottom, keeping sinus open until pocket is quite nearly filled in. Of course each treatment will require less and less gauze, and at no time after the operation should there be pus present; if there is then you have not thoroughly used your curette. I know that many of you will think that this method of treatment is severe and difficult, but I want to assure you that such is not the case, and the pain that I cause by such an operation is not at all severe. In treating several hundred of these cases I have only used a general anesthetic twice. To those that have not tried this method I ask them to try it and see if it does not prove helpful and a tooth saver. I recommend it most cordially over the old method of treating through root canal for months and then losing the tooth in the end.

CHAPTER XIII.

Infection, Instrument Sterilization, and Germicides.

Infection. Carrying Infection. A Germicidal Solution. Broach Sterilization. Instrumental Sterilization. Germicides; Some Dental Uses.

The subject of infection and instrument sterilization bears such an important relation to the treatment of pulpless teeth that I decide to include in this volume parts of two articles written by myself, the first on infection, published in *The Dental Summary* for June, 1903, and the latter published in *The Dental Review* for May, 1903.

Infection.

To infect means to introduce into the tissues a poison or virus which has the power of invading and multiplying, thereby setting up serious disturbances of physical well-being. This disturbance may be local only, or it may affect the whole organism as well; indeed, it is certain if the local infection is violent, that the system as a whole will suffer.

This is a subject upon which much has been written of late years, and yet I am of the opinion that the profession does not appreciate its importance. I have formed this opinion as a result of many years' experience in directing the treatment of cases in a large public infirmary, as well as in private practice, to both of which many serious cases are sent by regular practitioners. I think it is true that most dentists have learned to recognize syphilitic cases, and are fully aware of the danger of infecting themselves and others from them; also I believe that a large majority understand the precautions that are necessary in order to avoid doing so, and is it not a fact that a majority of dentists regard this disease as the only source of danger? I am certain they practice as if they do. This article is written to point out some of the most common ways in which we may infect pulps, gums or other tissues of the mouth, and to suggest a method to avoid doing so. Infection depends upon certain things, among which are:

First—The nature and condition of germs infecting, with reference to virulency.

Second—The condition of the part through which infection occurs, with reference to location, cellular elements of the tissues as well as its chemistry.

Third—The condition of the system as a whole, with reference to nutrition, including where the physiological functions are disturbed, infection takes place more readily.

All agree that the mouth is usually a "hot-bed" of micro-organic life; almost every known variety there abound. Were is not for the fact that the saliva is normally antiseptic to a slight degree, and is constantly moving, we would have more difficulty in getting open wounds in the mouth to heal. In this sense nature favors healing. When tissue becomes injured, abraded or loses its vital force, and infectious material is confined within or retained upon it for a period of time, of course the liability of infection increases; for these reasons I want to call attention to the advisability of thoroughly cleansing surfaces through which hypodermic needles, lancets or exploring needles are to be introduced. Surgeons quite universally take these precautions, but how many dentists do? How many are in the habit of forcing needles or lancets through surfaces that are covered with all sorts of infectious material? When pain, soreness and sloughing follow, we wonder why. Before scaling teeth, how many take the precaution to first rid the mouth of all putrefactive material lying upon the gum margins around the necks of the teeth?

When you think of it, how important it is. How many take the same precautions before placing the rubber dam clamp around the necks of teeth? In the majority of cases that instrument injures the gum, and with rubber dam, retains the infectious material in contact with it for the length of time required for the operation. The same is true of rubber dam when applied alone or with ligatures, but to a less degree. The danger is even greater when wedges or separators are used. A rule, then, I would like to advance is: Always cleanse the teeth and gums, especially around the gum margin, of all particles of food and putrefactive débris before beginning any operation. The most particular attention should be given the teeth in the immediate field of operation. For this purpose I first use a ball of cotton in the pliers, saturated with dioxygen and carrying a little fine powder. With this I scrub the teeth and gums; then I use the compression air atomizer and wash out the interproximate spaces with an antiseptic solution. I thoroughly flood the mouth, depending as much on the mechanical cleansing as on the antiseptic wash.

With this method you can feel reasonably certain that you have reduced the liability of infection to a minimum, and it only required a minute. In operating on teeth, especially where the pulp is involved, I take the additional precaution of washing off the teeth with alcohol after the rubber dam is in position.

Carrying Infection.

In scaling teeth it is important to keep the instrument in a good germicidal solution, and do not carry scalers from a pus pocket on one tooth to another tooth without first wiping off and dropping it into the

solution. I am thoroughly satisfied that many carry infection from such pockets to the healthy peridental membrane or other teeth by carelessness in this regard.

A Germicidal Solution.

As a germicidal solution I use sublamine, one in two hundred, and for the purpose of wiping off the scales I use small squares of chinosol gauze. I attribute much of my success in treating "pyorrhea alveolaris" to these precautions.

My brother, I want to interrogate you again; you see I am after you. How often do you take a nerve broach from a putrescent canal and carry it up into a pulp you have devitalized, or a clean canal, without sterilizing it? Take a case, we will say an upper first molar with three root canals, one putrescent or filled with pus, the others clean, perhaps made so by you; now, honestly, don't you frequently take your broach from the putrescent canal and carry it into others without first sterilizing it? Don't you sometimes take a broach from a pus-filled canal and carry it into the canal in a neighboring tooth where you are removing a non-infected pulp? How often do you wind cotton on a broach with unclean fingers, and carry this into the canals? Perhaps you don't, but the majority of practitioners do. Then they wonder why teeth become sore after the pulp is removed. Many think they sterilize their broach when they dip it into oil of cloves, cassia, or such agents, and leave it there a few seconds, but I assure you they don't.

Broach Sterilization.

For the purpose of getting some fairly accurate information regarding broach sterilization, I undertook a series of experiments. The method was this: I took small pieces of broaches, both steel iridio-platinum, and sterilized them by heat to redness. When cool, which only took a few seconds, they were carried into foul root canals, pus pockets, abscesses and ulcers, then dipped into various agents and left various lengths of time, after which they were removed, washed, and dropped into tubes containing beef bouillon culture media, and readings made from time to time for 96 hours. Test tubes were made from each case used, and microscopic comparisons frequently made. The length of time broaches were kept in the medicament was gradually increased until no cultures were obtained. The table of results follows, and is self-explanatory:

Time required to sterilize broach.		*Time required to sterilize broach.*	
Medicament.	Min.	Medicament.	Min.
Oil of Cloves	37	Trikresol	5
" Cassia	35	Creolin	5
" Sassafras	40	Sublamine, 1 in 200	2

Time Required to Sterilize Broach.

Medicament.	Min.
Oil of Peppermint	40
" Cade	25
" Birch tar	25
" Wintergreen	60
" Cajeput	30
" Cinnamon	40
" Eucalyptus	45
Carbolic acid	20
Creosote, Beechwood	25

Time Required to Sterilize Broach.

Medicament.	Min.
Chinosol, 10 per cent solution	1
Dioxygen	25
Bichloride of Mercury, 1 in 500.	10
Campho-phenique	20
Hydronaphthol, 20 per cent in alcohol	20
Beta-naphthol, 20 per cent in alcohol	32

These tables only prove so far as small broaches are concerned, but not for larger instruments, although they will readily show what agents would be likely to be most effective for such. They show the absurdity of attempting to sterilize an unclean broach by simply dipping it in any of the essential oils, or in fact any but a very few agents. Chinosol, ten per cent, proved most efficacious, but cannot be used for steel, as it corrodes it, but for other metals it is very valuable. Corrosive sublimate has the same objections. Carbolic acid, trikresol, and the like, are too irritant to be carried into soft tissue by instruments, and unless you wanted their special escharotic effects in a root canal, you would scarcely like to dip broaches in them. From my experience I recommend the use of sublamine, 1 to 200, for steel instruments; chinosol, 10 per cent, for those of other metals.

Instrument Sterilization.

The most practical method of sterilizing instruments after each case is by boiling water containing a slight amount of sodii bicarbonatis, for which purpose I have had made a small sterilizer consisting of a zinc box with a tight closing lid four inches wide, eight inches long and seven inches high, into which a removable tray is fitted and arranged so the instruments will stand upright. This box rests upon a standard and has a gas jet underneath. I had it made by a tinsmith at an expense of $7, and I think it fills the requirements better than any other. It does not heat the room to any extent, is sightly and very convenient, and inexpensive to run. If instruments are boiled in it for 15 minutes you can be reasonably sure they are well sterilized. The instruments we need to be most careful about sterilizing are clamps, separators, files, reamers, trimmers, scalers, lancets, needles, broaches. The simplest, easiest and surest way of doing so is by boiling. Before boiling they should be well scrubbed with soap and water. Try the precaution herein suggested and you will be delighted—at least they are very helpful to me.

Germicides: Some Dental Uses.

A germicide is an agent that destroys germ life and their spores. It is a term of recent origin, and is derived from the Latin, gérman = germ + cædere = to kill; literally, to kill germs. In dental literature the term is quite generally used to mean pus-germ destroyers. It is only since the germ theory of putrefaction became understood that this word, germicide, has taken on its present significance. The recent studies into the phenomena of life, physiological chemistry and pharmacology, bid fair to completely change our present system of therapeutics. We are beginning to see that our present accepted so-called rational system of treating pathological conditions is indeed most irrational and empirical. Not much longer will it do to treat certain conditions with certain remedies simply because our fathers did, or even because we have observed in a previous case good results followed like treatment we must now know the reason why.

There is no department of medicine (using the term medicine to include our and all other specialties) that is so unscientific as that of therapeutics. Enough work has been done to show conclusively that all remedial agents, of whatever nature, that have any action upon the physical organism do so by means of the chemical relation which they bear to the organ, tissue, or pathological condition treated. They act by means of a certain selective chemical affinity. Certain organs and tissues under certain conditions attract and appropriate certain medicinal agents, when so placed as to be accessible. Scientists have for several years recognized what is known as the chemotactic property of cell life—the attracting and repelling force which one cell or set of cells exert toward another. They look upon all organized life as a multiplication of cells, each having a specific function or functions, and each related to the other in a chemical way. The whole physical life process is a chemical one. The laws which govern the selection and preparation of food digestion, assimilation and throwing off waste material are chemical. This is not only true of the whole organism we call man, but is equally true in the micro-organic world. Furthermore, it also holds true in the relation of the former to the latter. The baneful influences of micro-organic life upon higher organisms is exerted through chemical processes. The solution of animal cell tissue, plastic exudate in wounds, and formation of pus are all chemical processes in which micro-organic life plays the important rôle.

Tonight I want to present the thought of destroying the micro-organic life and their baneful influences in animal tissue, by chemical means.

*Read before the Chicago Dental Society, Jan. 6, 1903.

The disassociation theory of Arrhenius, which had many able exponents, and which has been developed to a marvelous point in recent years, throws much light on this problem. The theory explained in a few words is this: When certain organic and inorganic acids or salts are carried into solution, either in the body or outside, they split up into ions—the negatively charged ones called anions, and the positive ones called kations. The action of such agents, therefore, depends upon the nature of its ions. This fact was brought out through a marvelously interesting series of experiments of Professor Jacques Loeb, formerly of the University of Chicago, but now of the University of California. No longer do we deal in the main with the molecules of which a substance is composed, but with the ions into which it breaks up. "We know, for example, that we can substitute at will sodium iodide for potassium iodide, in order to produce certain therapeutic effects. These salts are alike in that they both yield I-ions; they differ in that the former yield sodium ions and the latter potassium ions. Any similarity manifested in the therapeutic effects of these two salts is determined by the similarity of their iodine ions. But we know that the potassium iodide is much more depressant than the sodium salts. This is due to the direct poisonous effects of the potassium ions upon muscle and nerves, an effect not exhibited by sodium ions."*

This same principle holds true regarding the germicidal action of drugs. They are efficient in proportion to the number of ions they contain. In mercury compounds, for example, it is not the amount of mercury in the salt, but the number of mercury ions that determines the efficiency. Example: A given per cent solution of $HgCl_2$ in alcohol; a solvent in which slight disassociation occurs is less potent than aqueous solutions.

What is needed now is an extended study of the exact action of various ions. We must learn what kind of ions produce a certain result. Then the chemist will have little difficulty in furnishing us with substances capable of disassociating into such ions as we need for a given purpose. This disassociation may often be brought about by first undergoing some change or changes within the tissue, and then going into solution and disassociation by means of the solvent in the tissues. With these ideas in mind, the chemists have been at work with no end of new remedies as a result, many of which are useless because they have not been sufficiently tested, but rushed into the market to precede some other fellow. A few are excellent, and to some of which I want to call your attention. While these studies have been going on the physiologist has been at work, and shown us that germicides act upon the protoplasm

*Dr. Martin H. Fisher, *American Journal of Physiology*, 1901.

of the proteid molecule in this chemical way. Proteids are the most important substances occurring in animal and vegetable organisms. None of the phenomena characteristic of life occur without their presence; they are invariably and constantly constituents of protoplasm. They are highly complex and uncrystallizable (for the most part) compounds of carbon hydrogen, oxygen, nitrogen and sulphur. The difference between the proteid molecule of higher forms of multi-cellular life and that of the purely vegetative forms has not yet been well made out.

An enormous amount of work is necessary to bring out the exact relation and the exact composition of each. The inorganic salts, especially those of the heavy metals, such as mercury, iron, copper, lead, zinc, etc., act by forming insoluble compounds with protoplasm of bacteria. They do not penetrate deeply into the cell, and their action is, therefore, uncertain and usually very slight, $HgCl_2$ being the most potent of the group, because of its special toxic property, but its efficacy is greatly lessened if there are other proteids present, especially in the solutions which can be safely used on account of their toxicity.

The fatty acid series, the coal tar derivatives, phenol, naphthol, resorcin, salol, thymol, guaiacol, cresol, etc., and to this group we may add beechwood creosote, salicylic acid, etc., also act by coagulating the protoplasm to a greater or less degree; but with these agents the coagulum is quite soluble, and so the agents, if kept in contact, penetrate deeper, and to that extent are fairly germicidal, especially to germs that have an easily permeable cell wall, and this is especially true of carbolic acid, which is more or less volatile.

It must be understood that none of these agents acts in a chemical way, but simply by coagulation, which is a molecular process. None of these agents enters into chemic combinations with the proteid. While the salts of the metals produce insoluble precipitates, and thus prevent greater penetration, so that their germicidal power depends upon the degree of precipitability of the different proteids, the aromatic series, to which belong the essential oils, can scarcely be called germicides. They act by simple irritation; in no sense chemic.

The oxidizers and reducers all tend to produce chemic changes in micro-organisms. They all act rapidly, and are rapidly decomposed by all organic matter. Hydrogen dioxide is perhaps the best known of this class of agents. The rapid effervescence is evidence of its rapid action. The failure to get good germicidal results from this class lies in the difficulty to bring each germ into contact with the agents long enough to be destroyed. This difficulty is increased a hundred-fold when used within the tissues of the body, for the reason that they are equally active towards the organic matter of the tissues.

There is a fact which is often lost sight of in considering this subject which is of vast importance, and that is this: In the application of germicides to suppurations we must consider the tissue in which the suppurative process is going on. Nearly all these old agents act more forcefully against the cells of the tissue than against the micro-organisms therein.

Many, and indeed, most germicides are so coagulant, or otherwise destructive of the cell tissue, as to make their use in concentrated form dangerous, and, indeed, most of them possess general toxic or other deleterious properties after absorption which often endanger life. Therefore, in the practical applications of germicides, we must always consider:

1. Action on the system.
2. Action on the tissues of the part.
3. Action on the germs in the part.

And this brings us to two important points for consideration, namely, (1) the stimulating influence that certain agents exert toward the normal cell elements of the part; (2) the antiseptic influence that certain agents exert upon the whole organism, through the medium of the blood stream.

When suppurative micro-organisms get into the injured tissue of a part, by any means, there occur some interesting things. The injured tissue will soon be seen to be literally filled with reparative cells, cells which are carrying the necessary elements of repair to the injury, and carrying away the useless, discarded elements to be excreted and thrown off from the body. Mixed into this veritable beehive will be seen these micro-organisms, and if conditions are favorable they will grow and multiply rapidly. A "battle royal" occurs between these invading enemies and the reparative cells; sometimes one is victorious and sometimes the other, depending upon (1) the condition and nature of the micro-organisms; (2) the condition of the cells of the part; (3) the condition of the general system. There is some interesting detail in this connection, but time forbids further elucidation. It must, however, be clear to everyone, and this is the point I am trying to bring out, that favorable resolution may sometimes be brought about by directing our attention to any or all of these three things; (1) We may destroy or inhibit the growth of the micro-organism direct. (2) We may stimulate the cells of the part to increased activity and they in turn destroy, break down, these enemies. (3) We may act upon the whole organism with reference to stimulated circulation, assimilation and excretion, or increase the blood antisepsis, or any one or all of which, within certain limitation, would be equally potent so far as results are concerned. This explains why we have long been using certain agents which are not, strictly speaking, germicides, with good results. Iodoform, for example. I want

to emphasize, if I may, the need of attention to all three of these things, if we would be very successful in treating serious suppurations. In every serious infection we should always look to the nature of the micro-organism infecting; the condition of tissues of the part; and the condition of the whole system, with reference to nutrition, including excretion and circulation, and also the condition of the nervous system, before we determine what agent or agents we shall use.

The methods employed for determining the germicidal power of agents are many, all of which are imperfect, and whenever you read a statement of the germicidal power of any agent you must know the nature of the germs used in the test; how they were previously grown; how they were tested; in what media they were grown before and after, and what was the method of subjecting them to the agent, before you can have any idea of its value. All tests only prove so far as these things are known, and do not prove anything beyond that; because an agent proves germicidal toward a particular germ or mixture of germs, under certain conditions, using any method, only proves so far as that series, but does not prove anything so far as other germs or methods of using are concerned; therefore *all* experimental tests are only relatively valuable, and only useful for comparison, and beyond that prove nothing.

The literature of the medical and dental professions is full of conflicting statements regarding the potency of various agents, classed as germicides, the reasons for which are explained by the foregoing statement. In most cases I have succeeded in duplicating their experiments when the above conditions have all been stated. In not a few instances I have clearly demonstrated their faulty technique. I have tried almost every published method at some time or other in the last five years, and have concluded that the method suggested by myself in 1899 is open to the least objection, and yields results most nearly uniform, and yet I do not wish to convey the idea that this method will any way accurately tell what will occur when applied to actual practice in treating suppurations in the living tissue; but when these results are applied to such treatment, and there studied, and modified to meet condition, good results will follow. Until the chemistry of the proteid molecule under its various pathological changes is more clearly made out this is the best we can do. Pharmacology, the study of the action of remedies when practically applied, must at present be our main reliance. Science and experience must go hand in hand.

In making experimental tests, it is essential that the agent used be pure and reliable; that the germs be exposed to it in equal numbers under the same conditions; that they be at their maximum height of virulency, should be pure cultures, and that they be cultivated in media and tem-

perature most favorable to their growth. In the experiments from which the following tables were made up the following method was used; Organisms were grown in bouillon made from lean beef (not beef extract) in the usual manner, and neutralized with sodium hydrate (not sodium bicarbonate). In series D and E it was made slightly alkaline. The germs were grown and distributed throughout the media in equal numbers, as shown by microscopic examination. The germs were transferred in loopfuls to small squares (a centimeter) of filter paper, which was previously sterilized and kept in a petri dish; there they were allowed to dry; then on to this was carried by means of the loop sufficient of the medicament to completely cover the filter paper, and left for various lengths of time, when each square was washed, so as to remove the medicament, and planted in fresh tubes of culture media, and placed in an incubator, at 37° Centigrade. Readings were taken from time to time for a week. The germicidal power of the medicaments is here determined by the *time* necessary to expose germs to it, and, as you will see, a great difference appears. You will notice that some agents were used in full strength and others in per cent solutions, according as they could be used in practice.

In all of these series of experiments I began by exposing the germ to the medicament five minutes, and worked each way from that point, according as growth appeared or not. When doubt existed, inoculations were made in fresh media and in animals—guinea pigs and young rabbits mostly.

In these tables only final results are given. They are made up after many repetitions.

Series D.

Germ used, staphylococcus pyogenes aureus. Grown and plated out from abscess pus.

Agent.	Per Cent Solution.	Time Required, Minutes.
Oil cassia	Full strength	55
Oil cinnamon	Full strength	55
Oil cloves	Full strength	55
Oil cajeput	Full strength	50
Oil eucalyptus	Full strength	60
Oil wintergreen	Full strength	60
Oil peppermint	Full strength	55
Oil cade	Full strength	50
Oil birch tar	Full strength	30
Oil pennyroyal	Full strength	42

Agent.	Per Cent Solution.	Time Required, Minutes.
Carbolic acid	95 per cent	30
Creosote, B. W	Full strength	40
Campho-phenique	Full strength	40
Guaiacol	Full strength	40
Thymol	Alkaline, Saturate solution	30
Thiocol	Alcoholic, Saturate solution	30
Aspirin	Alcoholic, 9 per cent solution	22
Bichloride mercury	1-1,000	20
Phecene	Sat. solution	12
Creolin	Full strength	3
Trikresol	Full strength	5
Sublamine	1 in 250	5
Kresamin	Full strength	5
Phenol sulphonic	Full strength	5
Formalin	Full strength	3
Chinosol	10 per cent solution	1

Series E.

Germ, streptococcus pyogenes in virulent form from periosteal abscess.

Agent.	Per Cent Solution.	Time Required, Minutes.
Oil cassia	Full strength	60
Oil cinnamon	Full strength	60
Oil cloves	Full strength	60
Oil cajeput	Full strength	55
Oil eucalyptus	Full strength	60
Oil wintergreen	Full strength	60
Oil peppermint	Full strength	55
Oil cade	Full strength	40
Oil birch tar	Full strength	30
Oil pennyroyal	Full strength	35
Carbolic acid	95 per cent	30
Creosote, B. W	Full strength	40
Campho-phenique	Full strength	60
Thymol	Alkaline, Saturate solution	40
Thiocol	Alcoholic, Saturate solution	32
Aspirin	Alcohol, 9 per cent solution	22
Mercury bichloride	1 in 1,000	15
Phecene	Sat. solution	10

Agent.	Per Cent Solution.	Time Required, Minutes.
Creolin	Full strength	5
Trikresol	Full strength	5
Sublamine	1 in 250	5
Kresamin	Full strength	5
Formalin	Full strength	3
Chinosol	10 per cent	1

Series F.

Germ, Proteus bacillus.

Agent.	Per Cent Solution.	Time Required, Minutes.
Oil cassia	Full strength	55
Oil cinnamon	Full strength	40
Oil cloves	Full strength	45
Oil cajeput	Full strength	50
Oil eucalyptus	Full strength	50
Oil wintergreen	Full strength	55
Oil peppermint	Full strength	50
Oil cade	Full strength	40
Oil birch tar	Full strength	30
Carbolic acid	95 per cent	20
Creosote	Full strength	15
Thymol	Liquor. potass., Sat. solution	20
Thiocol	Alcoholic, Sat. solution	10
Aspirin	Alcoholic, 9 per cent solution	18
Naphtha eucalyptus	Alcoholic, Sat. solution	10
Chinosol	10 per cent solution	1
Mercury bichloride	1-1,000	22
Phecene	Sat. solution	10
Creolin	Full strength	8
Trikresol	Full strength	5
Formalin	Full strength	1
Tribromo phenol	Alcoholic, Sat. solution	8
Trichlorphenol	Alcoholic, Sat. solution	8

Series G.

Germ used, mixed pus culture.

Agent.	Per Cent Solution.	Time Required, Minutes.
Oil cassia	Full strength	40
Oil cinnamon	Full strength	40
Oil cloves	Full strength	40

Agent.	Per Cent Solution.	Time Required, Minutes.
Oil cajeput	Full strength	45
Oil eucalyptus	Full strength	40
Oil wintergreen	Full strength	60
Oil peppermint	Full strength	50
Oil cade	Full strength	25
Oil birch tar	Full strength	20
Oil pennyroyal	Full strength	45
Carbolic acid	Full strength	30
Creosote, B. W	Full strength	30
Campho-phenique	Full strength	40
Mercury bichloride	1-1,000	25
Creolin	Full strength	5
Trikresol	Full strength	5
Sublamine	1 in 250	3
Kresamin	Full strength	5
Formalin	Full strength	2
Chinosol	10 per cent	1
Phenol sulphonic	Full strength	5
Tribromo phenol	Alcoholic, Sat. solution	10
Trichlorphenol	Alcoholic, Sat. solution	8

Series H.

Bacillus pyoscyaneus. Isolated from pus.

Agent.	Per Cent Solution.	Time Required, Minutes.
Oil cassia	Full strength	38
Oil wintergreen	Full strength	45
Oil cinnamon	Full strength	40
Oil cloves	Full strength	40
Oil cajeput	Full strength	45
Oil eucalyptus	Full strength	40
Oil wintergreen	Full strength	40
Oil peppermint	Full strength	40
Oil pennyroyal	Full strength	40
Carbolic acid	95 per cent full strength	10
Creosote, B. W	Full strength	20
Oil sassafras	Full strength	40
Creolin	Full strength	5
Trikresol	Full strength	2
Formalin	Full strength	1

Agent.	Per Cent Solution.	Time Required, Minutes.
Sublamine	1 in 250	2
Bichloride of mercury	1 in 1,000	5
Kresamin	Full strength	3
Phenol sulphonic	Full strength	2
Chinosol	10 per cent strength	1
Campho-phenique	Full strength	10
Eugenol	Full strength	30
Permanganate of potash	10 per cent strength	30

Series 7.

Germ, bacillus prodigiosus.

Agent.	Per Cent. Solution.	Time Required, Minutes.
Oil cassia	Full strength	35
Oil cinnamon	Full strength	35
Oil cloves	Full strength	35
Eugenol	Full strength	32
Oil cajeput	Full strength	40
Oil eucalyptus	Full strength	40
Oil wintergreen	Full strength	40
Oil peppermint	Full strength	30
Oil pennyroyal	Full strength	35
Carbolic acid	Full strength	15
Creosote	Full strength	18
Trikresol	Full strength	2
Kresamin	Full strength	2
Bichloride of mercury	1-1,000	5
Sublamine	1-500	2
Permanganate of potash	10 per cent	25
Phenol sulphonic	Full strength	5
Chinosol	10 per cent	1

These tables only show the time required to completely destroy all life. Nearly all agents showed marked restraint in less time. Many of the germs exposed to the essential oils fifteen, and, indeed, thirty minutes, grew as quickly and as luxuriantly as the controller.

You will note the excellent showing made by the following agents: Formalin, sublamine, phenol sulphonic acid, trikresol, creolin, kresamin, phecene, chinosol.

The application of germicides as such to treatment of oral diseases is quite limited. It is only in violent, acute, chronic, necrotic suppurations;

in syphilitic ulcers, eczema, etc., and in each case the selection of the particular agent will be determined by the conditions present. They are also of value as hand and instrument disinfectors.

Formalin is a colorless liquid, resembling water in appearance, and is a 40 per cent solution of formaldehyde gas. It is probably the most potent germicide that can be used. Its dental uses are limited, because of its extreme irritating property. I have used it in old chronic abscesses, but in nearly every instance severe pain and swelling resulted, which has caused me to abandon it except in weak dilutions in such agents as creosote.

Paraform, a new solid polymer, has recently been recommended. There is a class of cases where it is of value, if used with care. I refer to old blind abscesses on the roots of teeth, containing small tortuous canals. This agent readily gives up formaldehyde gas, which is very penetrating. It should only be placed in the large entrance to the pulp chamber, and not down in the root canals, and even then it stirs up some irritation. In coming to this conclusion, I have lost some teeth from its use, but if you are careful and use it as stated you will find it of excellent value. Recently some practitioners have recommended it as a component part of root fillings. I am somewhat skeptical of the result. In old chronic cases, where there is little or no discharge of pus, but instead a thin ichorous fluid comes weeping down into the canal, cases that are not causing any great amount of pain, but sore and constantly annoying, in all such cases I get good results from this drug. It always increases the soreness and inflammation, which soon terminates in resolution. Perhaps the most valuable use we can make of this agent is as a disinfectant for foul rooms, for operating rooms, where serious surgical cases are attended to; for instruments, especially those used on syphilitic cases.

Paraform has recently been put upon the market in tablet form, especially designed for use in Schering's sterilizer. It is both effective and convenient.

Bichloride of mercury as a germicide was first brought to the attention of the medical profession by Koch, since which time its use has become almost universal. It is a potent germicide toward all germs that have a very permeable cell wall. It is very corrosive, producing insoluble coagulum, and therefore limiting its power. Its most serious objections are its irritant and toxic properties. In dental practice its use has been quite generally abandoned, except as a hand disinfectant, and on gauze for packing suppurating antrum; also in syphilitic cases.

Sublamine. Ethylenediamine sulphate of mercury. A new agent, recommended as a substitute for bichloride of mercury.

Ethylenediamine is an organic base, with a chemical formula of

$$\begin{array}{l} CH_2 - NH_2 \\ | \\ CH_2 - NH_2 \end{array}$$

It is a clear, colorless liquid of alkaline reaction, and gives off the odor of ammonia. This substance is used in connection with several coagulant germicides, for the purpose of reducing their irritant property and increasing their penetrative power. Sublamine comes to us in solid form, and is freely soluble in water. I have been using it in the strength of 1 in 500, and find it but very slightly irritating. It is a non-coagulant, and will penetrate much more deeply than bichloride. In all the tests it has proven much more efficacious than bichloride, and certainly is much more agreeable to use. I can most heartily recommend it for sterilizing hands, washing indolent ulcers, flushing the antrum, for washing through chronic abscesses, sterilizing the skin before operations. For all these purposes I have been using it in my private practice, as well as in the public infirmary. It is a chemical germicide, carrying pus into solution.

Phenol sulphonic acid is a light reddish-colored liquid, made by combining equal parts of sulphuric and carbolic acid. It is not so coagulant or irritating as either of the substances from which it is made. It can be used in full strength for burning through old chronic abscesses, and is especially recommended where cases are of long-standing, with more or less of bone absorption around the apex of the root. It is valuable to enlarge root canals, and to burn out the socket after a badly abscessed tooth is removed. Of course, it must always be used with caution. A 50 per cent solution is especially recommended to aid in the exfoliation of necrosed bone; it will also disintegrate and dissolve small pieces of tooth and necrosed bone that may be left after burring or curetting about the jaws. I use it on gauze for the first packing after such operations, especially in the antrum. Weaker solutions may be used to wash out after surgical operations on the jaws. I have come to look upon it as one of my most valuable agents.

Trikresol; another product from Schering's chemical laboratory. Its composition is as follows: Ortho-cresol, 35 per cent; metacresol, 40 per cent, and para-cresol, 40 per cent. It is a clear liquid pungent odor, resembling phenol; turns slightly red on exposure to strong sunlight. Is soluble in 2 per cent water, but freely in alcohol and oils. It is a splendid germicide, as shown by these experiments, and an agreeable preparation to use. I have been using it about four years, and now find I am using it in almost every condition where I formerly used carbolic acid or creosote. It is not so escharotic as carbolic acid; will penetrate much

deeper into vegetable cells, and will destroy spores. Two per cent solution is antiseptic. I recommend it to burn out old abscesses; as a dressing in root canals in acute apical pericementitis; in putrescent pulp; to relieve odontalgia, applied warm or almost hot. It penetrates the dentine as readily as the essential oils, but does not discolor it. A little (not an excess) is useful as a dressing after pulps are extirpated, before filling root canals. To keep scalers and such instruments sterile while using, I keep them in a ten per cent solution in alcohol and water. It is an excellent agent, used full strength, as a first treatment in pus pockets about the roots of teeth.

Kresamin is the name given to a combination of ethylenediamine and trikresol, containing equal parts of each. It is a reddish-colored, phenol-like liquid; has an agreeable odor, and is very slightly irritant. It is practically non-caustic, and but feebly coagulant. It is powerfully germicidal, equal to 1 in 500 bichloride of mercury. I have been using it lately in the clinic, with most flattering results. I am satisfied if used in acute or recent chronic abscesses it will be of value. I wash through such abscesses freely with it. I have passed some around among a few of my dental friends, and they are all delighted with the results they are getting with it. It is freely soluble, and may be used as an antiseptic in dilute solutions. It is a chemical disinfectant. When brought in contact with thick pus, kresamin seems to immediately dissolve it, and turn it a dark brown color. In apical pericementitis from any cause, it seems to be of great value; also applied to inflamed pulps it has an immediate quieting influence. In all inflammations accompanied with pus formation I am sure of its efficacy.

Chinosol has a chemical formula of $C_9H_6No\ KSO_4$, and is prepared by the action of sulphate of potassium on chinoline, a basic coal tar derivative. It occurs in the form of a crystalline yellow powder, possessing a very slight odor, and pungent coal tar taste. It is freely soluble in water, but insoluble in ether or alcohol. It is a chemical germicide. When brought in contact with pus in the slightly alkaline fluid of the tissues, it is readily broken up into oxychlorine, and it is this that is so powerfully germicidal. So far as my knowledge goes, it is the most potent germicide, so far as pus germs are concerned, of any agent at our command. I began using and recommending it for the eradication of pus in 1893, since which time it is my main standby. It has two slight objections, namely, it corrodes steel instruments (not others), and has a slight tendency to darken teeth; such discoloration is very readily removed with any oxygen bleacher. Being almost devoid of odor, it is not a very good deodorant for foul-smelling dentine, but as a pus-destroyer it certainly has no equal.

It is practically non-irritating; is wholly non-coagulant and non-caustic. I use it in 2 per cent solution for washing out bad pus pockets, abscesses in antrum, and alveolus. I use a ten per cent solution in chronic, foul, violent abscesses, and all other violent suppurations. It is used only for the purpose of getting rid of pus. When you need to burn out necrotic tissue, it is not recommended. It is wholly non-toxic, and can be used *ad libitum* in these solutions. Injected into a forming abscess, boil or carbuncle, it will immediately get rid of the pus. In any case of violent pus infection, where there is danger of serious results, this is the most efficient agent; especially in streptococcus infection, which is active, you can use no better drug. Chinosol gauze, absorbent cotton, and soap may be had in the market. If you have an abscessed antrum, where pus is rapidly being formed, try chinosol irrigation, and chinosol gauze, pack to control it, and you will be delighted.

I have shown you these cultures and tests, and called your special attention to these agents, not because they are the only good ones, but rather because I have had the most practical experience with them. I have used all of them on many hundred cases in the infirmary, and have the written history of treatments to corroborate all I have said. Some of you I know have tried some or all of these agents; others have not tried any. I simply want to make a plea for their value over the remedies you are using. Won't you try them?

CHAPTER XIV.

Management of Discolored Teeth.

General Considerations. Causes of Tooth Discoloration. Methods of Tooth Bleaching. Agents. The Direct Oxygen Method. Method of Using. Ethereal Solution. Sodium Dioxide. Chlorin Method.

General Considerations.

There is no subject in the whole range of dental science that needs to be more thoroughly understood than the management of discolored teeth. The operator must always keep in mind the possibility of pulpless teeth becoming discolored and particularly is it important to avoid such results in the anterior part of the mouth. We should not use rusty instruments or medicines that tend to discolor the dentine and in a general way keep our cases clean and free from outside contamination.

The tendency of all pulpless teeth is to change their normal color. Not all pulpless teeth discolor perceptibly, but they all lose their normal translucency. In many teeth this change is not sufficient to attract attention, but upon very close examination it will be seen. It must not be taken as a positive proof that a tooth has lost its pulp when it is a slightly different shade in color from its neighbor; indeed, teeth of different denominations in the same mouth differ in this respect.

The cuspid teeth are always darker than the incisors or bicuspids, and in not a few instances one central will differ from the other a shade or more. I have had a few cases where discoloration was marked, and yet upon opening the chambers the pulps were found vital and healthy. I think it is an established fact that under certain inflammatory influences pigmentation of the dentine may result, and yet the pulp return to health, although the instances are rare.

Causes of Tooth Discoloration.

For convenience of study I like to group the causes of tooth discoloration under three heads.

First. Discoloration following the death of the pulp where there is no pulp exposure.

Second. Discolorations following death of pulp and subsequent exposure of the chamber to the secretions of the mouth.

Third. Special discolorations from the staining influences of medicines, amalgam fillings, etc.

In the first class the source of pigmentation is in the decomposition of the pulp tissue, and such other secretions as may enter the canals from

the blood stream and surrounding tissues entering through the foramen. In the simplest form it is the direct result of severe inflammations of the pulp, which result in the destruction of that organ. These inflammations may be the result of irritation of fillings, arsenic, traumatic injuries of all sorts, in which the vascular supply is the prominent factor.

The various changes produced in the pulp by such forms of irritation have been fully explained in Chapter X, and all I wish to do here is to offer such explanations as are at hand as to the process by which such resulting discolorations are produced.

In such cases the discoloration is directly due to the breaking down of the corpuscular elements of the blood in which the hemoglobin is set free from the red corpuscles, and passes into solutions which readily infiltrate the tubuli of the dentin, giving the tooth a decidedly pinkish hue. Every operator can call to mind many such cases, following arsenical application, where the pink cast can readily be seen, especially when the eye is aided by transillumination. Teeth so discolored readily change unless the operator interferes, from pink to yellow, which gradually grows darker until brown and finally become a grayish black.

The violence of the pulpitis seems to have something to do with the degree and rapidity of the discoloration and hence, from this standpoint, it is inadvisable to apply arsenic to an already inflamed pulp. Kirk says: "In passing through its cycle of color changes hemoglobin undergoes several alterations in composition, during which a number of definite compounds are formed, each having marked chromogenic features. Of these composition products methemoglobin (brownish red), hemin (bluish black), hematin (dark brown or bluish black), and hematodin (orange), are best known."

It is doubtless true that this accounts for those discolorations resulting from inflammation and death of the pulp before putrefactive decomposition sets in, but, when that process has begun, there are other elements to consider and, before we can understand them, it is necessary to take into consideration the chemistry involved. The chemistry of the proteid molecules of all albuminous material, including pulp tissue, is not very well understood, but enough is known to furnish a rational explanation for tooth discoloration resulting from the decomposition of the pulp tissue. The important elements in the composition of the proteid substance of the pulp tissue are carbon, oxygen, hydrogen, nitrogen, phosphorus and sulphur; in the putrefactive decomposition of this tissue certain chemical compounds are formed, among which are carbon dioxid, ammonia, hydrogen sulphid, and water, none of which in themselves cause the discoloration, but when hydrogen sulphide is brought in contact with hemoglobin in solution in the presence of oxygen sulfo-methemoglobin is formed,

resulting in a certain amount of ferrous sulphide and other iron salts being formed, and it is these that furnish the green, brown, and black colors when forced into the tubuli—at least they play a very important part in tooth discoloration. In the second group of cases there are added factors both in the causation and modifying influences upon the resulting discoloration which must be considered.

When a pulp dies from exposure and putrefactive decomposition occurs in the presence of such oral secretions as may enter through such exposure, the process is modified greatly, and the character of the discoloration likewise changed.

The breaking down of the tissue, the changes in the vascular elements and subsequent putrefaction, are more rapid in these cases, and a ready way of escape for forming gases and salts makes deep discoloration less liable, and then the oral secretions may bring some substances that will enter the tubuli and thus modify the character of the color and make successful bleaching difficult. In the third group of cases are included those discolorations resulting from metallic and medicine stains, and are among the most difficult to handle.

Metallic salts are very apt to stain the tooth substance by their chemical reaction with the hydrogen sulphid with which dentine is saturated in putrefactive cases. Iron from rusty instruments, particularly when brought in contact with iodine and copper, producing those greenish stains, which after a time become black, that we often see from the use of copper alloy fillings, posts, dowels, screws, etc. These stains are difficult to remove.

Silver and mercury stains are usually black and are the direct result of certain combinations of metals in dental alloys, in which the silver and mercury are so acted upon by the secretions of the mouth, that slight amount of salts are formed which, in time, stain the dentine. The nitrate of silver, sometimes used as an obtundant, always stains the dentin black. These are easily removed.

Methods of Tooth Bleaching.

The process of tooth bleaching is a chemical problem in which there must be a reaction between the agents used and the staining or discoloring substance.

The chemical reaction must result in the formation of such new compounds with the coloring substance as are either freely soluble and can be washed out, or colorless compounds which are stable and not liable to change back to their original state or into some other coloring substance.

If the chemistry of the coloring substance could always be understood it would be a comparatively easy matter to find a substance that would combine with it in such a way as to bring about the desired result, but at

the present time we find many teeth discolored by some agent the nature of which we do not know, and, therefore, some cases do not yield to any bleaching method at our command.

Agents.

There are two general groups of agents used at the present time for tooth bleaching.

First. Oxydizing agents, substances which give off oxygen in a nascent condition.

Second. Reducing agents, substances which have a strong affinity for oxygen.

In the first group, there are two distinct classes, namely: Direct oxydizers, such as hydrogen, dioxid and sodium dioxide, and indirect oxydizers, such as chlorin, bromin.

In the group of reducing agents only one substance has been used to any extent, and that is sulphurous oxid.

In the use of all bleaching agents there are certain things which are fundamental, and success with any of them will depend on how thoroughly the detail is carried out.

First. All metallic fillings in the tooth to be bleached must be removed.

Second. The rubber dam must be securely applied to the discolored tooth only.

Third. The apical third of the root must be filled with gutta-percha.

Fourth. The pulp chamber must be completely opened, and as much dentin as can easily be removed without endangering the integrity of the tooth should be cut away. The chamber should be enlarged to include the thin points where the horns of the pulp were.

Fifth. The shape of the cavity in the teeth must be such as to permit of rapid closing so as to prevent the escape of the gases as much as possible. If it is not so naturally, then it must be made so by using gutta-percha.

Sixth. No metal instruments should be used in the work with agents that will readily corrode them, and thus produce a new staining substance.

Seventh. The bleaching process should be continued until the discolored tooth is a shade or two lighter than its neighbors, for the tendency of all cases is to go back a little.

Eighth. When the bleaching process is finished the chambers must be lined by some white substance upon which permanent filling can be built.

Ninth. Teeth that are checked badly or whose dentine is exposed to the fluids of the mouth are very liable to discolor again.

Tenth. Teeth that have been bleached should be temporarily filled for three months in order to ascertain if the result is permanent before the trouble and expense of permanent operations are undertaken.

The Direct Oxygen Method.

In bleaching discolored teeth with hydrogen dioxid, two solutions are used, namely, a 25 per cent aqueous, and a 25 per cent ethereal solution.

The methods are slightly different; the ordinary hydrogen dioxid obtainable in the market is a 3 per cent aqueous solution, which is not strong enough for our purpose as a bleacher.

Its strength can be increased by slowly and carefully evaporating some of the water; this is best accomplished as follows: Select a small porcelain evaporating dish which must be smooth and free from flaws; into this pour two ounces of hydrogen dioxid, then float it on a water bath, cover with loose paper to protect from dust, and slowly heat until the two ounces are reduced to one-quarter of an ounce, which usually takes about 45 minutes; this will give about a 25 per cent solution, which is the most desirable strength, and will keep in a colored bottle loosely corked for several days.

Method of Using.

When the chamber and cavity are ready to receive this bleaching agent it is applied on a loose roll of cotton into the chamber, and also the outside of the crown is moistened, then a draught of warm air is directed on the tooth, which will assist in the free liberation of oxygen. The agent is renewed every five minutes or so for about four applications, between each of which I dry the tooth with warm air. In the majority of cases half an hour will be all the time that is required for the bleaching of recently discolored cases, particularly teeth of a pinkish hue.

When the bleaching is completed the dentin should be thoroughly washed with warm distilled water. A convenient little rubber bag for catching the water used in flushing can be made of rubber dam with the aid of rubber cement. This bag or pocket can be so shaped as to admit of passing up on the lingual of the teeth, and held by the dam holder and the overflow carried away with the saliva ejector. It is sometimes necessary to repeat the bleaching operation once or twice a few days apart.

In my hands this has proven a very successful method.

Ethereal Solution.

In bleaching with the 25 per cent ethereal solution of hydrogen dioxid the procedure is exactly the same, with this addition, that in very stubborn cases it may be sealed in the chamber for 24 hours.

The best results seem to be attained from this solution when it is rendered slightly alkaline by the addition of a little sodium dioxid.

Sodium Dioxid $Na_2 O_2$.

Sodium dioxid is another compound that readily parts with its extra atom of oxygen, and only differs in its bleaching effect from hydrogen dioxid by the action of its by-product.

When sodium gives up its atom of oxygen it becomes sodium hydroxid Na_2O, which has decided saponifying action upon all vegetable and animal oils and fats, and a solvent action on animal tissue.

I have called attention to its value as a pulp canal cleanser after devitalization.

The tendency of this agent then, is not only to chemically change the coloring matter in the dentin of discolored teeth, but also to saponify and dissolve the contents of the tubuli.

Theoretically at least this is the ideal bleaching agent, because it removes more thoroughly than either agent the contents of the tubuli and consequently the normal tooth translucency is restored instead of the opaque whiteness which often follows other methods. For bleaching purposes it is used in two different ways, and each has its advocates.

It is mostly used in the form of a saturate solution in distilled water. This solution must be carefully made, as the tendency is on combining with water to lose its extra oxygen by the heat generated in the combination.

The solution is easily made by placing a small graduate glass containing half an ounce of distilled water in a large pan of broken ice; into this a tiny bit of fine powder is taken on a wooden spatula and gently shaken into the glass; this should be repeated every fifteen minutes until the solution assumes a milky appearance throughout, indicating that a saturate solution has been obtained. In a few minutes the cloudiness will disappear, when the solution is ready for use. The method of using this solution is exactly the same as already described for hydrogen dioxid except that instead of cotton, asbestos wool should be used as a means of applying. The solution should be allowed to remain for about five minutes, when the dentin should again be washed and thoroughly dried, then another application and so on for three or four times, and while the dentin is thus saturated.

A 10 per cent solution of sulphuric acid is applied to neutralize the alkali and liberate hydrogen dioxid, which aids in still further bleaching When the desired point has been reached the dentin should be thoroughly washed with warm distilled water the same as when the former method has been used.

Another method of using sodium dioxid has recently been suggested. It consists of placing the sodium dioxid powder into the chamber, and to this adding distilled water, then wash, dry and repeat, finally flooding the chamber with 10 per cent sulphuric acid and washing the dentin as above described.

Chlorin Method.

The chlorin method has been used longer than any other.

It consists in liberating chlorin gas in the pulp chamber which in turn unites with the hydrogen of the coloring substance and thus destroying its color. The agent is obtained by decomposing calcium hypochlorite (chlorinated lime), in the pulp chamber by the action of dilute acetic acid. Another method of using chlorin is to pack the chamber with chlorin of aluminum and decomposing the same with hydrogen dioxid, in which case both chlorin and oxygen are given off—and it is probable that the oxygen is the bleacher. In the chlorin method no metallic instruments of any kind should be used, even gold is readily acted upon by chlorin and a yellow insoluble stain results.

When the bleaching powder is placed in the chamber, and the other agent applied, the opening should be instantly closed with gutta-percha in order to prevent the ready escape of the chlorin, and in very bad cases the seal may be left 24 hours and then repeated if necessary. Many other agents and methods have been tried. At one time some operators were very enthusiastic over the cataphoric method, but none of these at all approached the direct oxygen method, and especially do I regard the sodium dioxid as the nearly ideal.

When the discoloration is due to metallic stains it is best to use direct chlorin method and follow with an ammoniacal solution of hydrogen dioxid. In a general way, it should be said that where one method fails, others should be tried, and even by so doing a few teeth will present where little can be accomplished in the way of bleaching.

The best lining for pulp chamber is paraffin where that can be used; it gives the natural translucent effect, which nothing else does. I make a solution in petroleum ether, and coat the wall toward the labial, when conditions will permit, and then fill the canal and chamber with white oxychloride of zinc, which has the tendency to continue the bleaching process. Before concluding this chapter let us repeat a few important points.

Gutta-percha must be the root filling for bleaching cases. The gum must be carefully protected else wide destruction will be the result; cover exposed tubuli if you expect permanent results. It is possible to carry the bleaching process so far as to destroy the integrity of tooth substance.

CHAPTER XV.

Diseases of the Peridental Membrane Having Their Beginning at the Gingivus.

Calcic Inflammation. Salivary Calculus. Treatment. Removal of Salivary Calculus. Scalers. Removal of Stains from the Teeth. Serumal Calculus. Phagedenic Pericementitis. Etiology of Phagedenic Pericementitis. Treatment of Serumal Calcic Inflammation and Phagedenic Pericementitis Diagnosis. Treatment. Instruments. Prognosis. Management of Loose Teeth.

In Chapter XII. we studied those diseases of the peridental membrane affecting its apical portion, and also reviewed its histology, to which the reader is asked to refer before beginning a study of this chapter, for the reason that the subject matter presented here can best be understood with the histology fresh in mind. The reader is asked especially to note the arrangement of the fibers at the gingivus.

The peridental membrane is subject to a great variety of diseases, many of which are little understood. Some of these diseases have their origin in the membrane, and others in the immediate surrounding parts. These diseases seem to become more and more prevalent with advancing civilization; and yet we have convincing evidence that the prehistoric races suffered from some of these diseases to a degree.

The prevalence of these diseases is partly accounted for by the fact that human teeth were designed to chew coarse foods, and as our habits have changed in this regard, so the teeth, gums and jaws have deteriorated. Organs that are not used atrophy. Then again, the excursion of coarse, tough foods over the teeth and gums tends to clear them of accumulating mucous and débris, and thus help in the preservation of the health of these organs. Perhaps the outdoor life of our ancestors, with its consequent robust health, had something to do with the health of the organs of mastication. Be that as it may, the facts are that more teeth are lost from these diseases than all other causes combined.

For many years these diseases, in a general way, were known to exist, and spoken of as one disease. Dr. J. M. Riggs, of Hartford, Conn., was one of the first to call the attention of the profession to this trouble, and outlined a kind of treatment in 1875, hence the term Riggs disease was given to it; other names that were applied to it were spongy gums, scurvy of the gums, diseased gums, inflammation of the gums, gingivitis, and latterly, pyorrhea alveolaris.

This latter term has been quite generally accepted by the profession as the most expressive of the conditions; it signifies a copious discharge of pus from the alveolus, which is very often present in these cases, but not always. If the term could be restricted to those cases which it describes I can see no objection to it.

About the year 1887 Dr. G. V. Black presented a classification of these diseases that seemed to more nearly describe conditions found, and yet there are cases that do not come within this classification. Dr. Black divides these diseases into three classes: Simple Gingivitis, Calcic Inflammation and Phagedenic Pericementitis.

Simple Gingivitis: By the term simple gingivitis is meant those inflammations of the gum which are not the result of calculus irritation. The simplest form of gingivitis we meet with is seen in young people, and occasionally where the teeth are clean. The gums become red and swollen, and bleed upon the slightest touch; the margin may be slightly thickened and everted, although the inflammation is not usually of a destructive nature. It is usually transcient, caused by some slight constitutional disturbance, and readily subsides when the teeth are kept clean.

A solution of resorcin used around the free margin of the gum, a fresh fruit diet recommended, supplemented by a slightly astringent and antiseptic mouth-wash, is usually all that is needed.

In others this inflammation may extend so as to include the entire gum septum and alveolar border. In these cases the gum is usually of a purple hue, and swollen sometimes so as to completely cover one or more crown surfaces of a tooth, or several teeth. Gums in this condition are more or less painful, and always bleed on the slightest touch. Fig. 45.

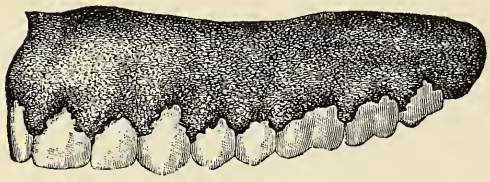

Fig. 45.
Showing hypertrophy of gum gingivus. (Burchard.)

Treatment consists of cleaning the toothneck and scraping the alveolar border where exposed, free blood letting and using powerful astringent applications such as iodide of zinc crystals, sulphate of copper, alum, glycerite of tannin.

Treatment should be daily at first, and then every third day supplemented with massage and vigorous brushing, and the use of astringent mouth-wash several times daily. There is another form of simple gingiv-

itis which seems to affect the gum tissue at the lingual or labial surface of upper incisors, labial surface cuspids and lingual of first molars.

The gum at first swells, and then drops away, leaving the root exposed for a considerable distance above the enamel line. There is no pus or tendency to bleeding, and all that can be done is to cleanse the surface by frequent massage, and instruction should be given the patient in the proper use of the brush, so as to keep the parts clean, and not force the gum farther away.

The most severe forms of gingivitis occurring where there are no deposits are constitutional in origin, and are usually, but not always, the result of the presence in the system of such drugs as phosphorus, mercury, lead and iodine, which are excreted by the fluids of the mouth, particularly the mucous and peridental membrane fluids. This will be considered in the next chapter.

There is another variety which I regard as the result of poor eliminations, auto-intoxication, in which all the secretions of the mouth are foul, and the gums hypertrophied and very tender; teeth are sore and the tongue badly coated. This can only be cured by proper physical treatment under the physician's direction, and all that is needed of the dentist is to cleanse the teeth and mucous surfaces, provide the patient with a suitable mouth-wash and otherwise meet the local conditions by such treatment as each case may require.

It is impossible to enumerate all the kinds of cases that come under this class, but I think sufficient has been said to point to the kind of treatment needed for all. I am quite sure that simple gingivitis often leads to more serious diseases of the peridental membrane itself. When the gingivæ becomes swollen, from any cause, and there is an accumulation of foul mucous and débris, it affords a good lodgment for bacteria. The gums soon become sore, teeth tender and consequently are not kept clean, because of the pain and the bleeding upon brushing. In most inflammations of this kind the patients will not use the teeth for masticating, and therefore the gums do not have the benefit of that natural method of cleaning, and this is another reason why they become more tender and inflamed. Such conditions also favor deposit of calculus. When calculus is present this rapidly passes into calcic inflammation.

Calcic Inflammation.

By the term calcic inflammation I mean inflammation of the gums and the peridental membrane, caused and maintained by the presence of calculus on the necks of the teeth. This is one of the most serious of all diseases of the peridental membrane. The degree of inflammation is dependent upon the amount and nature of the deposits. The greater

number of these cases I see leads me to conclude that some of these deposits are much more irritant than others. Cases occasionally present with large quantities of calculus on the crowns and around the necks, with little or no accompanying inflammation; and then again others will show the most violent inflammation resulting from a very small amount of calculus.

The calculus is of two varieties, named with reference to the source from which they come. These two forms occur separately or together; one above the other or mixed throughout.

Salivary Calculus.

The form most commonly found is known as salivary calculus, and is deposited by the saliva. These deposits are composed of earthy matter, consisting of saliva mucous, animal matter and phosphate of calcium, and perhaps other salts. It is usually most thickly deposited on those teeth situated nearest the openings of the ducts of the salivary glands, namely, the upper first molar and lower incisors and cuspids. It is deposited just above the gum or occasionally extending slightly under the gum margin. The amount of this sort of deposit on the teeth seems to depend largely upon two things: First, personal uncleanliness and carelessness about thoroughly brushing the teeth; second, upon certain constitutional disturbances. This form of calculus is most readily deposited at night, when the tongue and saliva are quiet, and hence the need of a most thorough brushing of the teeth upon arising. If taken thus early most of it can be brushed off before it has thoroughly hardened. Especially is this true if a stiff, properly shaped brush is used in connection with some good tooth powder. From this standpoint, the most important time to brush the teeth is upon arising in the morning.

As to the constitutional cause, I can say but little. We have not yet found out all there is to be known in this direction, but certain it is that there are some systemic conditions that favor these deposits. One individual who may be quite careless about brushing his teeth may have little or no deposits, and someone else who may be very particular in this regard, may be troubled very annoyingly. All that we know to do that will aid the system is to prescribe large quantities of fresh fruit juices, to be taken especially before retiring, also the drinking of large quantities of water.

In these cases of salivary calcic inflammation as fast as the peridental membrane is destroyed, the gum recedes, and the space is soon covered with fresh deposits; this process goes on gradually until after a time the alveolar border is absorbed, and the tooth appears to have grown up out of the socket until it becomes very unsightly. Oftentimes the gum sep-

tum is destroyed, and the entire alveolus disappears, and the tooth drops out. Whenever these deposits occur they destroy the tissue, and then deposit fresh calculus on the ground gained. The membrane is not destroyed to any great extent in advance of the deposits.

Occasionally, I see cases where the salivary calculus seems to deposit over the edge of the gums in great, thick masses, covering nearly the whole tooth crown. Particularly does this often occur with lower incisors. See Fig. 46.

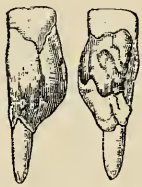

Fig. 46.
Deposits of salivary calculus. (Barrett.)

Treatment.

The treatment necessary for the relief of calcic inflammation, when due to the salivary variety, is very simple. It is always well to scrub the teeth and gums with hydrogen dioxide on a large ball of cotton carried in the pliers, the first thing; next, syringe around the teeth with 1-2-3 water, to remove as much infectious material as possible, and render everything as nearly antiseptic as possible. These precautions will often prevent severe inflammation following the cleansing of the teeth.

The essential thing in the management of all calcic inflammations is the thorough removal of the deposits, which is a simple matter when only the salivary variety is present.

Removal of Salivary Calculus.

There are two general plans of procedure followed in the removal of salivary deposits, one known as the *push method,* and the other known as the pull method. In the push method the general shape of the scaler is that of a chisel, the edge of which is brought to bear on the deposit at a point nearest the occlusal surface, forcing the deposit away with a chiseling motion. In the pull method the scaler is hooked at the lowest point of the deposit, just under the free margin of the gum, forcing the deposit off by pulling toward the occlusal. Each of these methods has its advocates, and the method easiest for one operator may not be for another, and perhaps the great majority of operators use both methods, as seems most convenient for the different locations.

In the use of either method it is essential that the scaler be grasped firmly, and the guide finger be firmly braced on the occlusal surface, or the occluso-labial or buccal angle, of the neighboring teeth particularly, is the essential when the push motion is used, in order to prevent accidents and serious injury to the gums or peridental membrane. Where the deposit requires considerable force for its dislodgment, patients are fearful lest the instrument slip and injure them seriously, especially when the push motion is used. The operator should always take pains to allay such apprehension; first, by verbal assurances, and second, by firmly bracing the guide finger.

The operator should always arrange the patient in such positions in the chair as will enable him to get at the various locations in the mouth in the most accessible manner. The mirror should be used to reflect light, and at the same time hold back the cheek, lips and tongue, as may be required from time to time. The operator should stand firmly on both feet, and have nothing around that will interfere with his free movement about the chair, and he should learn to assume as comfortable a position as possible, for there is nothing so fatiguing and eventually so ill-health-producing as standing for hours in strained, unnatural positions, which, if we allow ourselves to assume, soon become a habit, from which it is difficult to break away.

In scaling the lower teeth, the operating chair should be at the low position, patient in upright position, and the operator free to move about from side to side, sometimes assuming a position in front or right of patient, then to right back of patient, and left front, according as each position will bring in direct view the tooth surface upon which he is working.

Scalers.

The scalers which I find admirably adapted for the purpose intended are illustrated in Fig. 47. For several reasons, it seems best to begin the removal of salivary calculus on the lingual surfaces of the lower incisors first. At that point the deposit is thickest, and consequently a good impression is made at the outset on the mind of the patient as to the desirability and necessity of having the scaling done, and also because most easily removed at this point the patient gets over the fear or dread of the operation, before the more difficult places are undertaken.

Scaler Number 10 is intended to remove the heavy mass of the deposit in this position, and Number 9, in addition, to go in between the teeth better, and Numbers 5 and 6, being right and left push, are used to complement the other two, and especially to finish up after the larger part has been removed with 9 and 10; 7 and 8 are used to scrape around under the

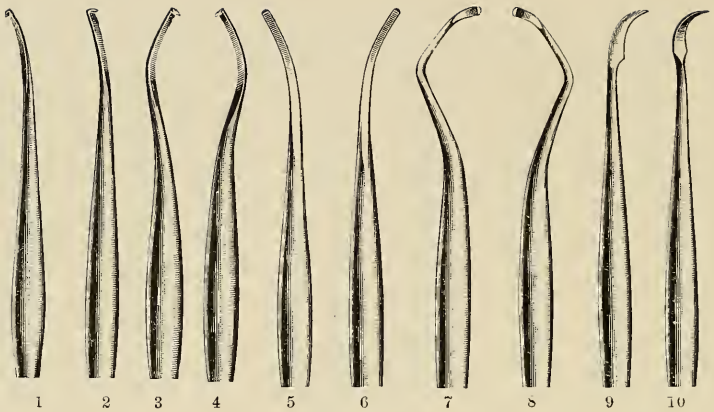

Fig. 47.
MaWhinney scalers.

gum margin, to remove any particles left by the other instruments. In the use of these instruments the rest finger is braced on the adjacent teeth, and with firm, positive strokes the deposit is dislodged, always holding the mirror in the left hand, with which the tongue is held back and light reflected on the field of operation. In removing deposits on the lingual surfaces of all the lower teeth, the author finds the right front position most convenient, occasionally changing to left front, if teeth incline out of normal position.

After the lingual surface and mesio-lingual and disto-lingual angles, are thus cleaned, using 1 and 2, I proceed to the labial and buccal surfaces and angles in exactly the same manner, passing from the incisors to the third molar on the right, then left, cleaning one tooth at a time as I go. As I get to the posterior part of the mouth I use 3 and 4 instead of 1 and 2, although Number 2 is well adapted to remove the calculus from the mesial surface of bicuspids and molars. I should also repeat here that warm antiseptic solutions in the syringe with which to wash away all loosened calculus must be at hand, and frequently used. I like this plan better than that of allowing the patient to take the solution from a glass, which consumes much time unnecessarily, although a glass of antiseptic solution should be at hand for the patient to use in case conditions arise requiring it.

The operator should always be careful that chips of tartar do not fly into his eyes. I have known two very serious accidents which occurred in that way.

In scaling the upper teeth the chair should be raised, and the patient's head thrown well back, and the front position taken, turning the head so as to bring each surface of each tooth into direct view. I begin at the

upper right third molar distal surface, passing around to the buccal, using Number 4; then from distal to lingual, using Number 3, and on the mesial, using Number 2. I proceed in exactly the same manner with the second and first molar, but with the bicuspids and incisors I use Number 1 for the distal. When I get to the median line I take the left third molar, using Number 3 for the distal and buccal surfaces, and Number 4 for the lingual, and pass along, completely cleaning one tooth at a time, until the median line is again reached, using 7 and 8 for finishing up, the same as directed for the lower teeth.

After all the teeth have been thoroughly scaled they should be polished, to remove all stains.

Removal of Stains from the Teeth.

For the purpose of polishing the crown surfaces of the teeth, many useful appliances have been devised, the best of which are in the form of rubber cups, wheels, cones, moose-hide points, brushes and wood points, all of which are to be used in the engine, and carry pulverized pumice, moistened with hydrogen dioxid.

Care must be observed not to lacerate the gum or burn the tissues, which is a thing likely to occur unless appliances of proper shape are selected. The rubber cup is usually most convenient for polishing around the gum margin, and the brush for other surfaces. As a final touch, the approximal surfaces should be polished with the flattened point of a rose-wood stick, carrying the pumice.

When the polishing is complete, silk floss should be passed between all the teeth to remove any tartar or pumice that may have lodged there, and the whole gum margins flushed with warm antiseptic solutions in the syringe, to wash out every particle of pumice, and finally the patient should rinse the mouth thoroughly with a palatable antiseptic solution. The patient is now ready to receive such instruction as to the proper care of his teeth as his case may require, and further appointments made, if other attention is necessary.

Serumal Calculus.

Many different names have been given to these deposits, most common of which are sanguinary deposits, gouty deposits, black tartar and serumal calculus. The last term seems to be most generally accepted.

Serumal calculus has its source in the blood stream, and probably directly from the serum of the blood that exudes through the tissues about the teeth. It seems quite certain that diseased conditions about the gum margins favor the deposition of serumal calculus. Probably all of the various forms of gingivitis already alluded to act as exciting causes. These deposits often occur on teeth in mouths kept scrupulously clean.

From a clinical standpoint, there does not appear to be much difference in different specimens of this variety of calculus.

Dr. Pierce held to the idea that serumal calculus contained large quantities of urates and uric acid, and that its presence in calculus was largely a result of the presence in the system of unusual quantities of urates and uric acid, and that gouty, rheumatic and albuminuric individuals suffered mostly. This idea was and is also held by Magitôt, Truman, Darby, Kirk, Marshall, Reese. On the other hand, Talbot states that only 6 per cent of a thousand specimens of different cases examined were found to contain uric acid or urates in any form, and claims the serumal calculus is deposited as a result of disease conditions present in the alveolus surrounding the tooth.

The great objection I see to the uric acid theory is that many individuals suffer from this kind of calcic inflammation when there cannot be found any trace of gout or rheumatic tendencies, and no excess of urates can be found in the system; and again, many who suffer from gout and rheumatism in most severe acute and chronic forms do not have the least signs of calcic inflammation. Then, again, many severe cases get well permanently, when only local treatment is given.

Perhaps no investigator along this line has done so much work as Dr. Talbot. He claims that both serumal calcic inflammation and phagedenic pericementitis are slightly different manifestations of the same disease, and that its immediate seat is in the alveolar process, and not a disease of the peridental membrane. He has given the name interstitial gingivitis to this trouble.

Dr. Talbot's theory regarding the diseases which the profession includes in the term pyorrhea alveolaris is about as follows: "This disorder is a local inflammatory condition of the gums tending to accelerate their normal tendency to disappearance at certain period of stress, or involution, of which the changes produced by old age are a type. There are two great causes—exciting and predisposing. The exciting may be purely local, or a local expression of a constitutional state. The local causes assigned are acute inflammation of mucous membranes, catarrhal states, germs, fungi, irregular teeth, lactic acid, pocket disease, serumal calculi, uncleanliness and local degeneracy.

"The constitutional causes—heredity, constitutional disorders, excessive lime salt secretion, meat eating, nervous exhaustion, scorbutus, uric acid and auto-intoxication states."

In the light of much clinical experience, I can scarcely agree with all of Dr. Talbot's conclusions, particularly regarding those cases which are here classified as phagedenic pericementitis—of which I shall hereafter speak; but, in the main, his conclusions seem rational, and have only been

arrived at after years of careful research, and are therefore entitled to honest consideration. His book on this subject should be read.

This variety of calculus is usually very hard, of a dark brown or black color, and is most frequently deposited in a ring just underneath the free margin of the gums. There are no specially favorable teeth where this variety is found. It occurs on any and all teeth. If allowed to remain any length of time it produces severe inflammation of gum margin, and often destruction of the gum septum and alveolar border. See Fig. 48.

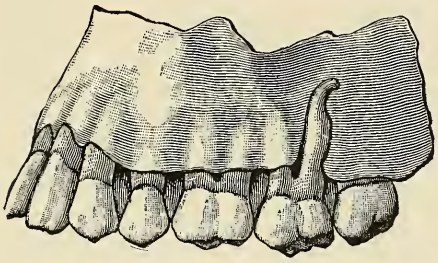

Fig. 48.
Showing absorption of gum gingivus and alveolus. (Burchard.)

Sometimes these deposits occur more on one side of a tooth than another, and pass rapidly toward the apex, covering one side of the root completely, in which case the gum septum will have disappeared, and the alveolus as well, to a considerable extent on that side, and the rest of the gum and peridental membrane remain normal.

There are other cases where the deposit occurs high up on the root, or perhaps between the roots of molars, and none to be seen around the gingivus, and, indeed, the membrane may seem to be attached all around the gingivus, and yet considerable serumal deposit be found high up on the root (see Figs. 49 and 50). Often there will appear a narrow, tor-

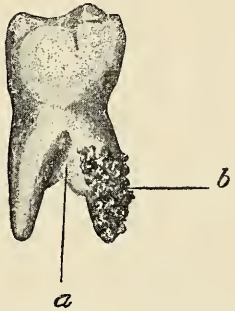

Fig. 49.
a, necrotic tissue; *b*, serumal calculus. (Barrett.)

tuous channel leading down to the gingivus, making the exact location of the deposit difficult to find; still other cases are seen where the deposit is high up on the root, and the alveolar wall immediately over it absorbed away, and perhaps a discharge of pus coming through the gum immediately over the deposit.

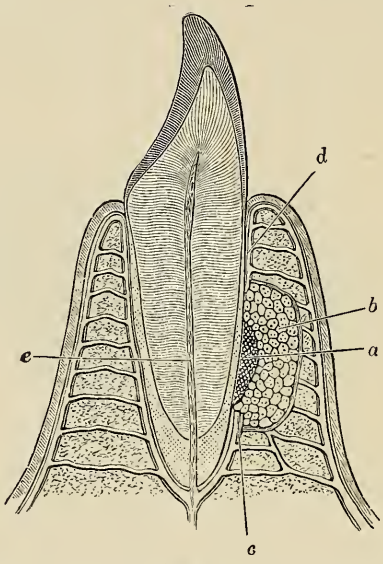

Fig. 50.

Serumal calcic inflammation. *A*, serum calculus; *b*, inflammatory corpuscles; *c*, *d*, peridental membrane intact; *e*, pulp. (Burchard.)

In some of these cases, where there is a large deposit of serumal calculus on one side of the tooth only, it is not unusual to find that the tooth is drawing away from its position; particularly is this true if the deposit extends high up toward the apex. Frequently this condition occurs between the central incisors. Indeed, I have seen such cases where the centrals have separated the width of a tooth. It is simply the effort of nature to get away from the irritant, and is partly due to the fact that the peridental membrane which holds the tooth in that direction is destroyed, allowing the remaining portion of the membrane to draw the tooth away toward the opposite side. In this case the gum septum may still be intact, although in most instances the septum of the alveolus is completely destroyed, and pus will be found flowing from the socket.

The gum and peridental membrane continue to recede, and in some cases salivary calculus is deposited above the original ring of serumal calculus, and this process goes on until the tooth becomes very loose in its socket. Sometimes these mixed deposits will encroach upon the apex, but more usually the deposit is of serumal variety. I have often seen

large deposits pass up on one side of the root until the apex was reached, resulting in the death of the pulp. Figs. 51, 52, 53.

In all of these cases the remaining peridental membrane becomes very much thickened and tougher, and in attempting to extract these teeth, after treatment has failed, you will be surprised at the force required to remove them. Oftentimes the membrane is so firmly attached to the gum

Fig. 51.
Serumal calculus covering the root of lower bicuspid.

Fig. 52.
Serumal calculus covering root of an upper cuspid, gum tissue was in normal position.

at some point as to result in badly tearing the tissues unless care be taken to cut it loose. I have frequently had cases where the teeth were so very loose that with the finger you could turn the apex out of the socket, but it was firmly attached on one surface by the peridental membrane, which made it necessary to clip off the gum on that side; otherwise I should have torn a great amount of gum tissue and periosteum in the operation. As a precautionary measure, it is well to pass a thin, sharp lancet around the neck of the tooth, and cut loose all attachment before removing these very loose teeth.

The important thing for the operator to remember regarding the etiology of this disease is that the immediate cause of calcic inflammation is the deposits present on the tooth root, and the first step in the treatment thereof is to thoroughly remove every particle of calculus, which, with this variety, is not always an easy task, but when successfully done, recovery in ordinary cases is very rapid.

Inasmuch as the scaling of teeth where serumal calculus is present does not differ from that necessary in phagedenic pericementitis, I shall

Fig. 53.
Serumal calculus covering root of molar.

reserve the description of that technique and subsequent treatment until after that subject is presented.

Phagedenic Pericementitis.

The term phagedenic pericementitis was first suggested by Dr. G. V. Black, in 1883, and while I cannot say it has been very generally accepted by the profession, yet it seems most expressive of the conditions present in this disease.

Phagedenic pericementitis is a destructive inflammation of the peridental membrane, which may or may not be accompanied by calcific deposits, and is nearly always accompanied with pus.

It usually has its beginning in some form of gingivitis, and most frequently starts at the gingivus and passes up along the root, destroying the peridental membrane as it progresses. Sometimes it passes along one side of the root in a narrow channel, which, toward the apical third, will widen out, forming a very distinct pocket. Other times it may affect all of one side of a root, either mesial, distal, labial or lingual, and I do not think it can be said that one side is more liable to be affected than another. The channel often takes a very tortuous course, and oftentimes the pocket will be found on one side of a root and the point of discharge or starting point at the gingivus on another side. In other cases there may be no pockets, but a progressive destruction of the entire membrane all around the tooth, but this form is not very common.

There are still other cases where the pockets seem to have no connection with the gingivus, but form high up on the root, with a discharge

of sero-pus through an opening in the gum directly over the pocket. Careful examination must be made in this class of cases, in order to avoid mistaking it for a chronic alveolar abscess dependent upon the death of the pulp. See Fig. 50.

In this class of cases the point of discharge differs somewhat from the fistulous opening or "gum boil" of an alveolar abscess, in which the discharge is through a small, conical, tit-like projection, with the bone underneath in fairly hard normal condition, while in this variety of phagedenic trouble the point of discharge is wide open, with gum tissue around it slightly elevated, thickened, very red, and will bleed on the slightest touch, and the bone underneath absorbed to considerable extent, so that an explorer will readily pass through the outer opening directly to the root, revealing a root denuded of pericementum over a large area, for in all these cases the pockets tend to spread laterally along the membrane, never forming pockets which lead away from the tooth to any extent, but are largest next to the tooth.

Many of these cases come to us while still in the acute form, and will present considerable soreness and show these raised areas on the gum over the root, which has never broken and discharged pus, and yet if you take a fine, sharp explorer, you will be able to pass it through this area directly to the root, and discover that much of the root has lost its membrane.

I have seen many cases of this kind, particularly on the labial surface of cuspids and buccal surfaces of upper molars and bicuspids, where, try as hard as I could, I was not able to find an opening at the gingivus leading to the pocket, and yet when I opened the pocket with a lancet I found roots bare to a considerable extent, in many cases extending from the gingival third to the apex, bringing about the death of the pulp, and oftentimes find such roots covered with serumal deposits, and many cases have no such deposits. Recently I had a case of a lady of about forty years of age, where two upper bicuspids had such pockets, involving the entire middle third of the roots, with small openings at the lingual gingival margin of each. The gum was in normal position, but the teeth were very loose, and digital pressure on the gum brought away great quantities of pus. The general health of the patient was such as to preclude any attempt at treatment, so the teeth were removed, bringing away the gingival third of the buccal alveolar plate, and revealing the fact that the middle third, and extending almost to the apex, had lost its membrane, and yet there were no deposits of any kind. This was a true phagedenic case in acute form, which had, as nearly as could be ascertained, been developed within three months' time. In my private practice, as well as in the infirmary of the school, I have seen many such cases,

but the percentage is small compared to those which are complicated with serumal deposits.

In many cases where the disease has progressed slowly, you may see a decidedly thickening of the bone, particularly at the gingivus, which is brought about by the mild stimulation of the osteoblast, resulting from continuous irritation. Usually the membrane has lost its attachment to the cementum, and the bone next to the tooth absorbed somewhat, and the thickening occurs by building on the outer surface of the bone.

Where these pockets exist, we occasionally see large absorption of tooth substance. I have collected several specimens. The absorbed areas present a variety of shapes, from broad, rough, shallow places, to small, round holes, that look almost as though they had been made with a drill. I am very glad to say, however, that these cases are rare.

With regard to the development and progress of phagedenic pericementitis, it should be said that cases present a wide difference.

Some cases begin and go on violently, until the tooth becomes so loose that it is lost within a few weeks; others progress very slowly, and take years to produce any serious trouble. In some cases the teeth early become very sore, sensitive to thermal changes, and the seat of neuralgic pains that become very severe. In other cases the development and progress of the trouble is painless, and the first indication of trouble is the looseness of the tooth; more particularly is this true of simple phagedenic cases, that is, those cases not complicated with serumal deposits. In such cases there sometimes is no apparent inflammation of the gums; on the contrary, they often appear anemic.

Etiology of Phagedenic Pericementitis.

All observers seem to agree as to the clinical aspect of these cases, but there is a wide difference of opinion regarding its etiology.

Dr. G. V. Black states that the disease is fundamentally one of the peridental membrane affecting its attachment to the tooth; fiber by fiber that membrane is torn away from the cementum, and is the result of specific infection, probably due to a special micro-organism which has not been isolated as yet, although he has done much work in an endeavor to find it.

Dr. George W. Cook is of the same opinion, and has done a great amount of work, resulting in finding a germ that he thinks is responsible for the trouble. Certain it is that he has produced similar pockets by introducing this germ under the gum, particularly where some irritation of the gingivus already existed.

Dr. M. L. Rhein presents some interesting theories. He calls this disease pyorrhea alveolaris, and divides it into two general classes, which he terms pyorrhea simplex and pyorrhea complex.

Under the head pyorrhea simplex he includes all those cases of what he calls purely local origin, and which are amenable to simple local treatment.

Pyorrhea complex he regards to be of constitutional origin, presenting slightly different forms, according to the physical disorder of the patient, which forms are readily recognizable.

This class he subdivides into four groups, according to their causation.

(a) Those due to disorders of nutrition, gout, diabetes, nephritis, scurvy, chlorosis, chronic rheumatism, anemia, leukemia, pregnancy.

(b) Those resulting from acute attacks of infectious diseases, among which he specified typhoid fever, tuberculosis, malaria, acute rheumatism, pleurisy, pericarditis, syphilis.

(c) Those due to nervous disorders, cerebral disease, spinal disease, neurasthenia, hysteria.

(d) Those conditions which are the result of drug poisoning, particularly mercury, lead and iodides.

It is doubtless true that many of these diseases do make a distinct impression in the mouth, but as a cause for pyorrhea alveolaris they can only be considered as predisposing and never as the exciting cause, because all forms of this disease are seen in the mouths of individuals who have never had any of the diseases enumerated, and, again, individuals who have suffered from many of these diseases have never had the slightest indication of peridental trouble; and, again, most forms of this trouble yield to local treatment.

Dr. Talbot's theories, which have already been explained, are to the effect that this disease has its seat in the alveolar process, to which I suggest the following criticism: If the process is the seat of trouble, why does the membrane lose its attachment to the tooth first? Many cases that I have examined show the membrane attached to the bone and torn away from the cementum. Again, why, on the removal of the affected teeth, does the disease terminate without further treatment? Why does the disease never occur in edentulous jaws?

Dr. J. W. Younger believes the disease to be purely local, and caused by a special germ. He states that from years of experience in managing these cases he very rarely finds one that will not yield completely to local treatment.

I have had several patients who continually suffered from simple phagedenic trouble requiring constant watching. No sooner would one

pocket be healed than the patient would return with one or more new pockets filled with pus, perhaps on teeth that had never been affected before. And after years of this constant watching, the trouble would entirely disappear following some severe illness, and in four cases has not reappeared in five years, and one case in ten years. Clearly, then, the changed physical condition had something to do with the disappearance of the disease.

I think I have collected enough opinions to show the reader that the mind of the profession is not made up regarding the etiology of this disease, but that there is a great difference of opinion regarding this point. I have faith to believe that soon new light will be thrown on this subject, for many able men are bending their best energy to the solution of this problem.

Treatment of Serumal Calcic Inflammation and Phagedenic Pericementitis.

Diagnosis.

The first step in the management of these cases is a correct diagnosis. We need to know exactly what the conditions are.

In making a diagnosis, first get a complete history of the case; find out about the character of the pain; how long has the trouble existed? Have any teeth been lost from this cause? Are the teeth sore to pressure; tender to hot and cold drinks; sweet and sour things? Are any teeth loose? Has there been any swelling? Is there an offensive odor and a bad taste in the mouth? Do the gums bleed readily?

A knowledge of these things gained from the patient not only is helpful to the operator, but has a good mental influence on the patient.

The instruments needed to make a thorough examination are two explorers, a mouth mirror, a mouth lamp, supplemented by a well-trained index finger. After learning the history of the case, a good way to make the mouth examination is to begin with a general survey of the entire mouth; then with a surgically clean index finger pass it along the gum high up, and then along the gingivus, and on labial, buccal and lingual surfaces of gum, using enough pressure to detect any soft, tender places, and to force any pus present out at the gingivus. Next press on the teeth to determine looseness and tenderness, and observe if any of the teeth have moved out of normal position; and, finally, with mouth lamp and mirror to light up every pocket, proceed with proper explorers to examine every surface of every tooth, passing the explorer around the free margin of the gum, dropping it into each pocket, noting its shape, and the character of the deposits. All the points observed should be

written down for future reference, and particularly should this be done if the case is sent to you for consultation.

Treatment.

The first essential in the treatment of these diseases is thorough cleaning of the teeth. Even in simple phagedenic cases, thorough scraping of the root at every point where the membrane has separated from the cementum is necessary, because there are always present either concretions of calculus or pus globules, or other foreign matter which must be removed.

Instruments.

The instruments necessary to do this work must be small, of good texture, and so shaped as to readily reach every surface of all the teeth roots in the mouth. Many sets of scalers have been devised for this purpose, each possessing certain merits. The Younger set of thirty-seven answers admirably for those who are skilled in their use, but I am satisfied they are too complicated for use of general practitioners, requiring years of constant using to become proficient. The Harlan and Cushing sets are used by many operators. The Tompkins set has excellent points.

The author's set (Fig. 47) was devised especially for students' use, and presents the following points:

1. The cutting edge is in line with the shaft, so the instrument does not roll in the fingers under stress.
2. The shapes are admirably adapted to reach every surface.
3. They are small, with rounded backs, and therefore injure the gum less than other sets.
4. They are stiff, and will not spring over hard deposits under stress.
5. They are easily sharpened.

In the main, the points that have been insisted upon for the removal of salivary calculus hold good in the removal of serumal deposits, to which the reader is referred for the instrumental technique. The important thing is to get it all off, which I can assure you is never an easy task. Always instruct your patient as to the conditions and necessary treatment.

The following outline of procedure should prove helpful:

First.—Clean the gums with hydrogen dioxid on a swab of cotton, and with an antiseptic solution in the syringe flush out all débris from around the necks of the teeth.

Second.—Keep the working end of the scalers immersed in an antiseptic solution.

Third.—Select some point in the mouth to begin on, and proceed from that, thoroughly cleansing one tooth at a time.

In this class of cases I begin with the lower right third molar, and pass around the lower jaw first, and then take the upper in the same way. The important thing is to take one tooth at a time and confine all your thought and energy to that one, until the scaling of it is completed.

Fourth.—When the pockets are deep and the tissues tender, I always pack the pockets with a rope of cotton saturated with one per cent solution of cocain in peppermint water, previously packing bibulous paper, gauze or absorbent cotton around the tooth, to prevent any of the cocain solution escaping into the throat. The cotton rope should be left in the pocket for two or three minutes.

Fifth.—If the tooth neck is very sensitive, I dry it and lay 30 per cent chloride of zinc around it for a few minutes.

Sixth.—If the pockets are tortuous and the gingivus tight around the tooth, I pack the gum away with a rope of zephyr wool, saturated in 25 per cent phenosulphonic acid, for twenty-four hours, which will not only gain ready access, but will tend to soften the deposits.

Seventh.—If the pocket is high up, the gum tight around the tooth neck, and the outer plate of the alveolus over the middle or apical third destroyed, I invariably make an opening through the gum at that point, through which to do the scaling.

Eighth.—Where the pocket is very large and membrane entirely destroyed at that point, it is well to curette the bone as well as the tooth, and treat as a surgical wound.

Ninth.—Use the explorer frequently to determine the progress made in scaling on each surface. Special care is needed in the grooves and depressions in root surfaces.

Tenth.—Flush the pockets frequently with antiseptic solutions, to keep a clean field upon which to work.

Eleventh.—Where the teeth are very loose some method of fixation must be adopted, for loose teeth never can recover unless held immovably.

Twelfth.—In very bad cases the pulp should be destroyed and canals filled. If pulp is dead, root canals must receive first attention.

The operator should be cautioned against attempting too much at one sitting, for not only does the patient become fatigued, but the operator loses that delicacy of touch which is so essential to successful work.

Sometimes a whole sitting will be consumed in scaling a single tooth, but one tooth well done is far better than more half done.

In very bad cases radical measures are necessary to bring about changed conditions. Dr. A. W. Harlan has said, in effect, that in some of these cases you need to tear up and literally burn the tissues involved

before recovery will result. Growth through stimulation by irritation seems to be the thought.

Most operators find the pull motion produces less pain, and is more positive in its results than the push motion. In the pull method the scaler, of proper shape, is carried to the bottom of the pocket first, and gradually worked toward the crown, removing and reinserting the scaler as few times as possible. I always use Numbers 7 and 8, which can be used with either a push or pull motion to finish up with. They are admirably adapted for reaching into grooves, crevices and all irregular surfaces.

In very bad cases, where the membrane is largely destroyed, many operators recommend extraction, cleansing, filling root canals, deepening the socket, and replanting. Oftentimes one root of a molar may be removed and the remaining one or two, as the case may be, carry the tooth successfully. These operations will be described in another chapter.

After the instrumentation has been completed, the pockets should be thoroughly washed to remove all débris and blood clot, which, if not removed, make a favorable field for the growth of micro-organisms.

I do not recommend hydrogen dioxid for this purpose, except in those cases where the pockets are wide open, for there is danger of the rapid effervescence tearing away the attached membrane.

As a first treatment, many operators use concentrated *lactic acid* for its irritant stimulating effect; others use 30 per cent chloride of zinc, 10 per cent trichloracetic acid, sulphate of copper powder, campho-phenique, Black's 1, 2, 3, resorcin, hydronaphthol, iodide of zinc, tincture of iodine, some of the silver salts, and a lot of other remedies have been suggested. In my hands a combination of iodine and iodide of zinc have proven very efficient for the ordinary case. This remedy was suggested by Dr. Talbot, and is made of five parts of iodine crystals; seven parts of iodide of zinc; glycerine to make thin cream. Sometimes, if the pockets are large and flow of pus profuse, I will burn it out with 50 per cent phenosulphonic acid, and pack it with chinosol gauze, which I leave for forty-eight hours. In the average case I dry the gum and pocket as well as I can, and flow on the gum, and into the pockets Talbot's glycerol iodine and zinc freely, keeping them dry with a piece of aseptic dental napkin for a couple of minutes, when the excess may be washed off with antiseptic solution in the syringe, and the mouth rinsed. The patient must return in twenty-four hours, when, if there is pus present, I feel certain that at that point the tooth has not been thoroughly cleansed, and I proceed to do so.

It is very rare, indeed, that pus presents at the third sitting.

If the case is progressing, I do not again open the pockets, but allow the same medicine to flow down by gravity, or capillary attraction. Of course, at each sitting the gum and necks of the teeth must be cleansed, for

which purpose I usually use a warm antiseptic solution in a compressed air atomizer.

I repeat this treatment every third day for a week or so; then every fourth day; then once a week, and so on, never dismissing a case under three months, except in rare instances.

I frequently change to plain tincture of iodine, especially if the gums get very sore from the iodine and zinc, and if the necks of the teeth are very sensitive I use chloride of zinc, nitrate of silver, or trichloracetic acid, as conditions indicate. The patient should always be provided with an antiseptic astringent mouth wash, and good tooth powder.

℞

Sodii boras,	ʒ iv
Acidi Carbol,	ʒ i
Glycerite Tannin,	ʒ ii
Tinct. Myrrh,	ʒ i
Ol. Cassia,	ʒ ss.
Ol. Peppermint,	m.x.
Alcoholis,	℥ ii
Aquæ dist., q. s. ad.	℥ vi

M. Sig. Use a teaspoonful in one-quarter glass of warm water as a mouth wash several times daily.

This should be supplemented by thorough brushing, using a good powder and a hard brush. Daily massaging the gums with the finger moistened with the above solution, directing the force toward the tooth crown, will also prove helpful in most cases. The patient's general health must be looked after; all the functions of the body must be normally performed. The eliminative organs should be stimulated. The daily drinking of eight glasses of pure water should be insisted upon, and a minimum amount of sweets and meats taken, and the patient should be encouraged to eat plenty of fresh, ripe fruit, and to take wholesome exercise in the open air.

In a work of this kind it is impossible to do more than indicate the line of treatment that should be followed, but the operator's knowledge of the conditions and the remedies at hand will be able to modify the treatment for each individual case.

A few things must be kept in mind. Perfect cleanliness, thoroughness as to detail. Insist on the patient doing his part, and do not tear open the pockets or poke instruments into them when healing has once favorably begun, and if there is any physical cause of irritation, such as improper contact points, rough filling margins, improper use of the brush or toothpicks, these must first be attended to.

Prognosis.

Regarding the prognosis of these cases I can only say that the cause which produced it in the first place will bring it back, and that no positive assurance can be given the patient as to freedom from future attacks, and the fact that such attacks recur is no proof that the disease was not cured in the previous attempt, for this disease is not like smallpox, in which one attack insures the patient against a future one.

Teeth in all varieties of this disease have been cured and remained so for many, many years. The fallacious notion given the public by many dentists that this disease is incurable is not only untrue, but it is unjust to the many scientific and skilled men who are making a success of the treatment of the great majority of cases presented, and equally unjust to those who suffer and lose their teeth because of this false idea.

Before dismissing this subject, a word should be said regarding the reattachment of the peridental membrane.

Does the peridental membrane reattach itself when once torn from the cementum? The answer must be in the affirmative, when the conditions are favorable. If the membrane is attached to the bone side, and only separated from the tooth in comparatively small areas, and the tooth in those areas has been thoroughly cleaned, the membrane will lie down on the cementum and reattach its fibres by a new deposit of cementum, but if the membrane is totally destroyed over any considerable area, reattachment is impossible, and only a mechanical union will exist between the bone and cementum at those points. For further consideration, see chapter on implantations, etc.

Management of Loose Teeth.

Many of these cases present with one or more teeth loose, and before permanent results can be expected the teeth must be held firmly in the socket. Oftentimes teeth are so loose as to make scaling impossible until they are held firmly in the socket. Many methods and appliances have been suggested for this purpose, most of which have for their object the binding of several teeth together in a solid compact. Splints of either rubber or gold are frequently made to fit along the lingual surfaces of teeth down to the free margin of the gum, and ligated in position by passing either gold wire or silk twist through small holes in the splint to the mesial and distal of each tooth, and tying on the labial or buccal.

Another method is to swage shallow caps to fit over the occlusal where the occlusion will permit. This may often be made in one piece to cover several teeth, and extending over the labial and lingual surfaces about two millimeters, and is cemented into place. (See Fig. 54).

Another method is to surround each tooth with narrow bands of gauge 26, 22 karat gold, allowing but one thickness of gold between the

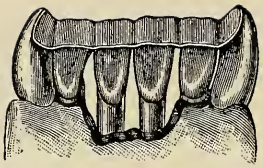

Fig. 54.
Swedged gold caps to retain lower incisors.

teeth. The bands should be on the occlusal third of the tooth, and held in position with cement. (See Fig. 55.) Many operators bind the teeth together with pure gold wire. (See Fig. 56).

Fig. 55.
Gold bands for retaining loose lower incisors.

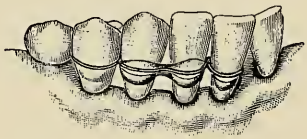

Fig. 56.
Showing wire ligature for retaining loose teeth. (Rhein.)

When it is only necessary to hold the teeth temporarily, I find no better way when teeth on either side of the loose ones are present than to ligate one to the other, using silk thread. Take a case where two lower centrals are loose. I begin by wrapping number A, waxed silk thread twice around the right lateral, tying on the mesial just below the contact point; pass it around the right central in exactly the same way, and tie on the mesial, and so on, around the left central and lateral. A little experience will enable the operator to adapt this same plan to various locations about the mouth.

Take this same case, where the teeth are very loose and the alveolus largely gone, a permanent appliance is necessary, and for this purpose I find nothing so good as to devitalize the pulps in the four incisors; grind the lingual surface slightly immediately over the opening made for

pulp removal and canal filling, over which burnish pure gold, each tooth separately, and pass a short post into each canal; take an impression with all in position, make a cast and flow solder over the whole, uniting all together, polish, and set with cement. I have several of these splints that have been worn from five to twelve years, with the utmost satisfaction.

The same method can be adapted to the upper incisors and cuspids, and oftentimes without devitalizing the pulps by making the hole to receive the pin to the mesio-lingual or disto-lingual of the pulp. (See Fig. 57.)

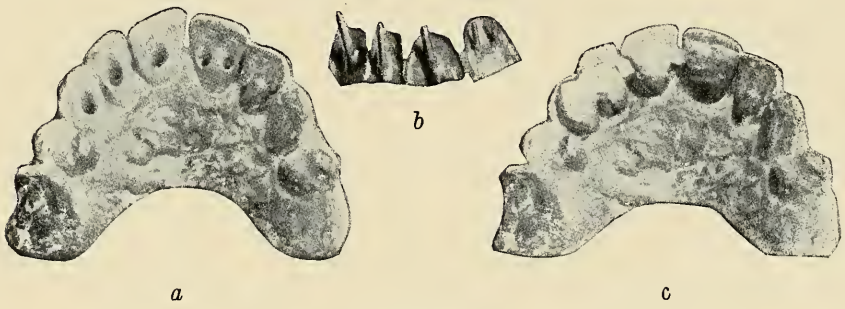

Fig. 57.
Splint for retaining loose teeth. (Ames.)

The same principle can be adapted to molar and bicuspids, using the occlusal instead of the lingual surface. Oftentimes by cutting a groove extending along the center of the occlusal from one tooth to the other, into which a heavy platino-iridium wire is laid, around which amalgam or gold foil is packed, a permanent result is obtained.

The Carmichael attachment is admirably adapted for this purpose.

When these badly diseased teeth are situated on either side of short edentulous spaces, good results are often obtained by cutting off and crowning the teeth, and bridging in the missing teeth. I have several such cases that have remained comfortable for ten years. It goes without saying that in all these cases the usual measure to effect a cure must be adopted, and carried out.

Before dismissing this subject, I wish to speak of a method suggested by Dr. W. V.-B. Ames, for the cure of the disease, and the retention of loose teeth, particularly lower incisors and cuspids that are very loose, and that have lost much of the alveolus surrounding them. It consists in destroying the pulps, filling pulp canals, and thoroughly cleaning the teeth, after which he saws the crowns off at the gum line and bands each root the same as for a Richmond crown, and in case the natural crowns are good, he utilizes them instead of porcelain for the crown, a detailed description of which does not come within the scope of this book, but which

can be found in the *Dental Cosmos,* May, 1903. Dr. Ames claims that by this method he not only fixes the teeth firmly in position, but the gold collar acts as a stimulant aiding recovery.

CHAPTER XVI.

Hypercementosis and Root Resorptions.

Morbid Anatomy. Causes of Hypercementosis. Pathology. Symptoms.

The terms Hypercementosis and Excementosis have been used synonymously to designate a secondary deposit of cementum on the tooth root; this deposit may occur upon any part or surface of the root, but is most frequent about the apical end.

In the normal process of tooth formation, cementum is deposited layer upon layer, which not only continues until the root has assumed complete form but slowly continues throughout life. Unless disturbed in the process of formation these layers pass in one continuous line from the enamel line to apex, differing very little as to thickness at different points until apex is reached, where it becomes slightly thicker and at the enamel junction it tapers off quite thin.

Morbid Anatomy.

It is difficult to say exactly where hypertrophy begins and the normal physiological formation ends. In ground specimens it is clearly seen that a certain layer often becomes greatly hypertrophied at a certain point and all the rest of it formed in normal thickness (See Fig. 58).

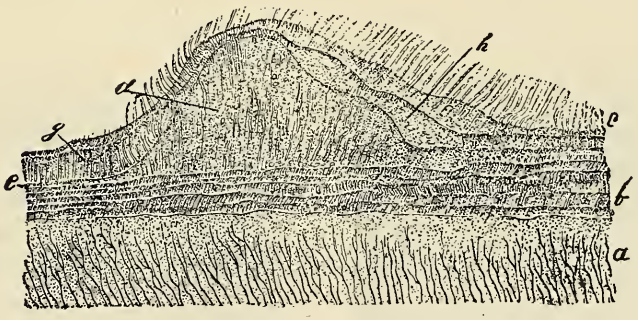

Fig. 58.

Hypercementosis. *A*, dentine; *b*, layers of cementum; *c*, peridental membrane; *d*, thickened layer; *e*, same layer normal. (Black.)

Hypercementosis may occur in one or more of these regular layers or lamellæ, and it may occur in the first layer or any or all subsequent layers, and in many ground specimens it is clearly seen that absorption of small areas of previously deposited cementum occurs, which is afterwards filled in as the next layers are formed.

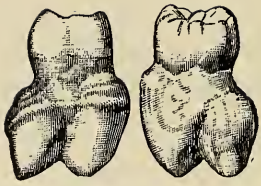

Fig. 60.
Hypercementosis of entire root. (Barrett.)

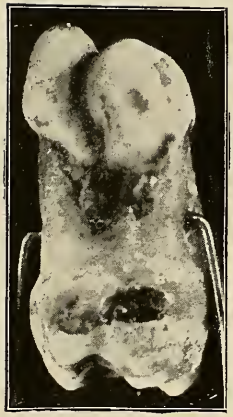

Fig. 61.
Hypercementosis, round, smooth form, involving apical third of root.

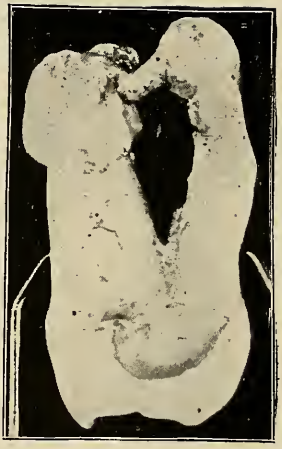

Fig. 62.
Hypercementosis, with deposit of cementum uniting root apices.

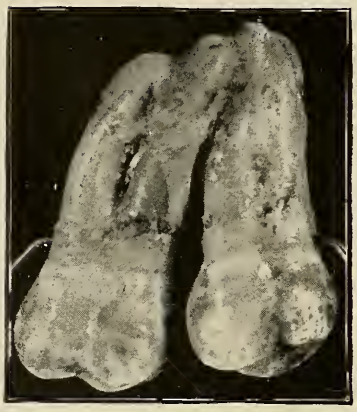

Fig. 63.
Hypercementosis, showing the union of roots of two molars.

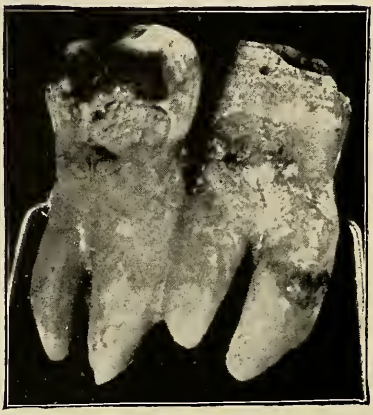

Fig. 64.
Hypercementosis. Union of distal root of lower second molar with mesial root of lower third molar.

Teeth affected with hypercementosis present a variety of forms. In some cases the entire root is enlarged (see Fig. 60) ; in others the enlargement is confined to the apical third, (Fig. 61,) and in still other cases the cementum may have united two roots of the same or adjoining teeth. (Figs. 62, 63, 64.)

The accompanying illustrations will furnish interesting study. They are made from specimens among the writer's collection. (Figs. 65, 66, 67, 68.)

Fig. 65.
Hypercementosis, irregular form.

Fig. 66.
Hypercementosis, round, smooth form, involving the apical third.

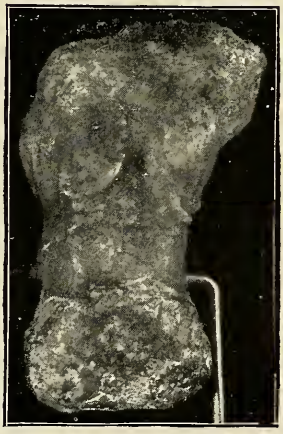

Fig. 67.
Hypercementosis, irregular form.

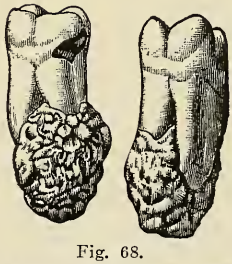

Fig. 68.
Calcific structureless mass involving roots of molar. (Barrett.)

Causes of Hypercementosis.

As to the causes of hypercementosis very little can be definitely stated. It is doubtless due to some undue irritation, which disturbs the normal physiological process. It seems probable that many times the irritant is chemical in its nature, and has its source in the blood of the surrounding parts, and in a great majority of cases is due to a mild form of irritation to the peridental membranes.

This irritation may be the result of continued extra stress on the tooth, difficulty in erupting and passing into normal position, and certain forms of chronic pericementitis.

In short anything that will cause hyperemic condition of the peridental membrane, and through that and over activity of the cementoblasts is liable to cause hypercementosis, but the facts are that a very small percentage of teeth subjected to unusual stress or irritation of every kind are ever affected with this disorder.

Pathology.

The pathology of hypercementosis is vague and uncertain, and many writers are of the opinion that it has no defined or distinct pathology.

The facts are that most of the cases that have come under my observation presented no untoward symptoms of any kind, and were only discovered when teeth were removed for other reasons.

A few writers report cases where hypercementosis seemed to be the cause of trigiminal neuralgias; shifting pains about the face, eyes and ears have been attributed to this cause, but evidence is wanting to prove such claims, and even when the extraction of such a tooth is followed by cessation of pain does not prove it to be the cause. See chapter on neuralgia.

Diagnosis.—A diagnosis can only be made by exclusion.

When every other possible cause of these disturbances has been examined into with negative results, hypercementosis may be suspected, but the only means of positively determining the existence or non-existence of hypercementosis is the X-ray.

Resorption of the roots of permanent teeth.—By this term is meant the absorption of portion of roots of permanent teeth.

As has been previously stated, cementum, and even dentin, is often absorbed in certain areas and refilled with new cementum, and this appears to be a purely physiological process.

The work of cutting out is doubtless done by osteoclasts, a multi-nucleated cell, which is situated here and there among the pericementum fibers. The work of building up is due to the cementoblasts which have been referred to in the beginning of Chapter XII.

Many writers are of the opinion that when absorption is going on at one point, an extra amount of cementum is being deposited at another point. At what point the absorption process can be called pathological I do not know, but it is my opinion that the entire process is physiological and is Nature's method of getting rid of foreign inharmonious bodies.

That sometimes this normal process becomes perverted is doubtless true, but as to why such is the case we do not know.

Cause.—That any mild irritant is likely to act as a cause, anything that would act as a special stimulus to the osteoclasts would doubtless cause resorption.

In these hollowed out places osteoclasts are found in great numbers but as to the how or why they are there we do not seem to know.

The process is exactly the same as that occurring in implanted teeth, but differs markedly from absorption of roots of temporary teeth.

Resorption occurring in permanent teeth seems to attack the cementum much more rapidly than the dentin. In all the specimens that I have seen even when the apex of the root was involved, the cementum was removed from large areas of dentin, showing that dentin is more resistant to its action, while if you examine a temporary undergoing this process you will find the dentin is hollowed out, undermining the cementum to considerable extent.

As to the pathology of resorption very little can be said as previously stated. This is Nature's method of getting rid of any aseptic foreign substance, which doubtless explains why planted teeth lose their roots by this process, because it must be quite clear to every one that a planted tooth, which has no peridental membrane for its support and nourishment can not be considered as anything but a foreign substance, although, of course, more in harmony with its surroundings than most anything else that could be put there.

This is Nature's method of getting rid of teeth, where their function is accomplished, but once in a while a temporary tooth fails to absorb, and insists on remaining even when the permanent one is ready to occupy its place.

Why this is so, we do not know; a good deal of guesswork has been indulged in regarding this whole subject, and very little fact adduced.

Symptoms.

What are the symptoms of root resorption?

I do not know of any except in cases of plantation when the tooth often has short periods of soreness, an itching feeling in the gums and bone, and a feeling of uncertainty which is cleared up when the tooth loosens and drops out.

Diagnosis.—In this case also a positive diagnosis can only be made by the X-ray.

Treatment.—Relieve the conditions that present, look to the root canal, and the peridental membrane and relieve any irritation that can be found and failing to relieve and make the tooth comfortable, extraction must be resorted to.

CHAPTER XVII.

Resection of Roots and Plantation of Teeth.

Replantation as a Cure for Alveolar Abscess.

The operation of resection consists in the excision and removal of a portion of any organ; and is especially applied to the end of bones and teeth. The resection of a root is an operation for the removal of its apical end, although the term is often applied to operation for the renewal of an entire root of a two or three rooted tooth to which the term amputation is commonly given.

Elsewhere I have alluded to the fact that frequently in persistent alveolar abscess the root apex is partially absorbed, resulting in very rough sharp surfaces, which interfere with the healing process. In such cases the operation of resection is advised especially when one of the ten anterior teeth on either upper or lower jaw is involved.

On the molar teeth I do not consider this operation practical except where the entire root is to be removed. The first step in resection after the root canal filling is completed is to make all the surrounding tissues surgically clean.

Next a circular incision is made in the gum over the root apex and the flap thus formed is desected up and held with a suitable instrument; the root is exposed by cutting away the bone with a suitable bur in the engine, oftentimes the bone has been absorbed to considerable extent making this part of the operation very simple. The parts should now be cleansed and bleeding stopped. With a bibevel drill a hole is made in the center of the root a little above the point at which it is desired to cut it off, Fig. 69; into this hole is introduced a fissure bur, the engine run rapidly cutting to right and left from the hole, thus cutting off the desired portion which can easily be removed with a small excavator.

The root end should be carefully rounded. (Fig. 69B.)

The entire pocket should be thoroughly curetted and washed out with antiseptic gauze. The irrigation and packing should be repeated every two or three days, using less gauze each time and watching that healing from the bottom occurs. This operation is not difficult, and with the aid of a local anesthetic can be done quite painlessly, and the results are quite satisfactory. I know of several teeth doing good service that were thus treated over six years ago.

Amputation.—The removal of the entire root of a molar tooth is to be recommended when that root has lost its membrane, and much of the

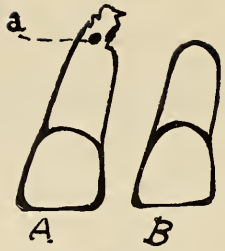

Fig. 69.
A diagram of tooth with apex absorption. a, the drill hole; B, the finished rounded tooth apex.

alveolus and the other root or roots are sufficiently healthy to insure the useful retention of the tooth.

In some severe phagedenic cases, especially when complicated with serumal calculus, we often find the lingual root of upper molars so badly diseased that recovery is impossible and yet the two buccal roots may be in a good state of attachment; in all such cases amputation of the lingual root is to be recommended.

This operation is very simple and is most easily done with a fissure bur in the engine, cutting off the root on a level with the bifurcation; the root can easily be removed with a small elevator or root forcep. A stone should then be used with which the tooth at this point should be beveled toward the occlusal and lingual surface so that no shoulder remains on which food will lodge (see Fig. 70).

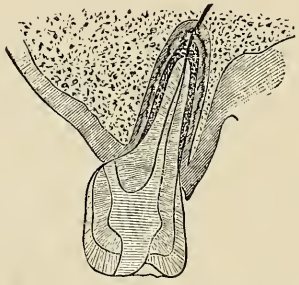

Fig. 70.
Showing upper molar with lingual root removed. (Black.)

The socket from which the root is removed should be curetted and kept clean until it fills in with bone, which will be an added protection to the remaining roots.

This operation is adapted to lower molars as well and in rare cases to two rooted bicuspids (see Fig. 71). The prognosis for such cases is

very good, a great majority of such teeth when the operation has been well done will do good service for many years. Indeed, I regard this operation most satisfactory of all the operations suggested in this chapter.

Plantation.—The plantation of teeth has been practiced for many years with varying success. The subject matter is best presented under three heads: Replantation, which is the replacing of a tooth in the socket from which it is removed; transplantation, which is the insertion of a natural tooth in a natural socket from which another was recently extracted and implantation, which is the insertion of a tooth root in a socket in the alveolus which has been artificially made.

The operation of replantation has been practiced for many years. As far back as I have access to the literature, I find dentists have recommended the immediate replacement of teeth that have been accidentally lost. Indeed mothers and fathers have frequently replaced teeth of their children that have been lost by accidental means such as blows, kicks of horses, falls, etc., especially, when a thread of attachment remained, and in many cases good results were obtained. These facts induced dentists to remove teeth that were hopelessly diseased either from alveolar abscess or pyorrhea trouble and replant them.

Replantation as a Cure for Alveolar Abscess.

In some chronic forms of alveolar abscess where there is considerable absorption of bone, replantation has been suggested as a cure and many successful cases are reported, but this practice has been quite generally abandoned in favor of the more successful method of curetting the bone around the root apex, and the root as well when foreign deposits are present or resection when the apex is absorbed or roughened.

Replantation as a cure for so-called pyorrhea alveolaris was practiced to a considerable extent a few years ago, but at the present time has fallen into disrepute, partly because of improved methods of treating the disease. The operation consisted in the extraction of the affected tooth, immersing it immediately in antiseptic solutions, under whch it is thoroughly cleansed of all foreign matter and if roughened it is made smooth.

The root canal is cleansed through the apical end unless a cavity exists, then the canal is quickly dried and filled with gutta-percha, and the tooth again immersed in the antiseptic solution, where it should remain until the socket is made ready. The socket is thoroughly curetted and slightly deepened, and the tooth inserted while fresh hemorrhage ensues.

The tooth must be immobilized by the use of bands, caps or wire ligatures as seems best adapted to the case. This practice has been generally abandoned for this class of cases because at best the alveolar process

around the tooth in bad pyorrhea cases has already been destroyed to a very considerable extent, and it is therefore quite impossible to establish a socket that will retain the tooth for any great length of time; I have had a few cases remain two years.

Replantation is best adapted to those cases where healthy teeth have been accidentally dislodged and which can be replaced within a short period of time after the accident.

When such cases can be attended to within a few hours of the accident, best results are obtained. In every case the root canal must be filled, and the whole tooth rendered antiseptic, and the socket cleansed of blood clot, and if possible a normal hemorrhage restored. The tooth is then forced to place and retained by some of the appliances previously suggested.

I have a case of this kind where two centrals have remained for eight years, and are still doing good service.

Transplantation.—When from any cause a tooth is lost from a comparatively good socket it may be replaced by another tooth.

There are several things in the way of this operation which relate to the difficulty of getting a freshly extracted tooth adapted as to size, shape and denomination, to fit the already existing socket and many a patient is reluctant about having some other person's tooth in his mouth, and there in some danger of transmitting disease.

Some operators recommend old, dry teeth, but I have not been successful in their use, and among my professional acquaintances I find the practice has been quite generally abandoned, although there might be exceptional circumstances where it could be recommended, but a freshly extracted root even if an artificial crown is necessary, will often give sufficient service to repay for the trouble.

Implantation.—Dr. Younger was the first dentist to give any prominence to this operation and I think he is justly entitled to the authorship of this operation as it is practised at the present time.

His first great recorded operation was in 1885, since which time he has probably performed it more than any other operator, and his success is probably due to his splendid skill.

In this operation a tooth is taken from another mouth and planted in a socket which is artificially made in the alveolus. The operation is intended to supply a missing tooth where one has failed to erupt or where it had been extracted some years before. Here as in other cases a freshly extracted tooth or root is to be desired.

The special instruments necessary with which to perform this operation are the Younger trephines illustrated (Fig. 72); Ottofy spiral knives (Fig. 74), and the Ottolengui reamers (Fig. 73), and a few long-shanked burs of different shapes (Fig. 75).

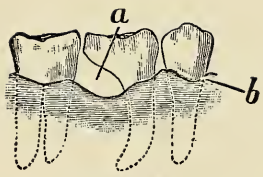

Fig. 71.
Showing lower molar with distal root removed. (Black.)

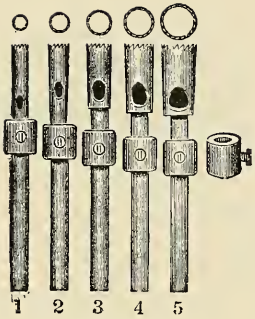

Fig. 72.
Younger trephines.

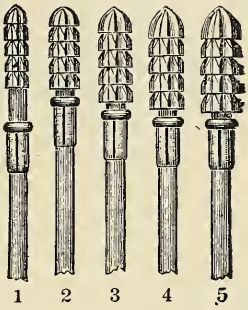

Fig. 73.
Ottolengui's reamers.

Fig. 74.
Ottofy's spiral knives.

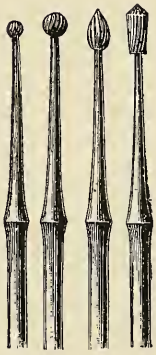

Fig. 75.
Long-shanked burs of necessary shapes.

The first step in this operation is to select a tooth that will fill the space accurately, and if a root an artificial crown must be properly adjusted both as to shape, form and contour.

The tooth should be kept in an antiseptic solution, and all the work done under antiseptic precautions.

Local anesthetics are all that are usually needed, although in many cases general anesthesia will have to be resorted to.

The writer follows the method outlined by Dr. Ottofy in 1887, which is as follows: A deep incision is made in the gum and periosteum at a point that will form the lingual gingivus (Fig. 76), and turned up and out toward the labial. (See Fig. 77).

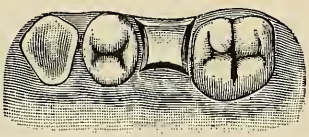

Fig. 76.
Showing Dr. Ottofy's incisions.

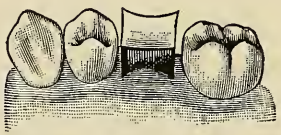

Fig. 77.
Showing the flap as suggested by Dr. Ottofy.

With proper trephines, knives and burs a socket is drilled to receive the new root. The tooth should be tried in the socket at frequent intervals to insure a proper fit, bearing in mind that the root must fit the socket very tightly. After the socket is drilled it must be cleared of blood clot and bone chips and a fresh hemorrhage started into which the root is forced and retained by a suitable appliance. Every effort should be made to keep the parts antiseptic until healing is complete.

General considerations.—Planted teeth are only held in the socket by the deposit of bone around the root and the union is only a mechanical one.

The peridental membrane is never again formed or reattached except in very rare instances where teeth have been immediately returned to other sockets when only partially knocked out.

Dr. Younger and many others have held that there is a rejuvenation and reattachment of the membrane, but careful investigation does not substantiate their claims.

However, it must be said that such teeth often become more firm in the jaw, and require greater force to dislodge them than their neighbors, which is accounted for by the fact that the newly formed bone is more dense, and has not the natural channels through it that are present in the normal alveolus.

When planted teeth are lost it is usually by process of absorption. This absorption sometimes begins at the apex and gradually proceeds crownward, but more often it begins on the sides of the root; this process

sometimes ceases for a time and bone is deposited in these places, but sooner or later all these teeth will be lost, quite after the plan of absorption of temporary teeth.

The operations of transplantation and implantation should be confined to the incisors, cuspids, and bicuspids, and should only be attempted where it seems very undesirable to replace the missing teeth with artificial ones either in the form of plates removable or fixed bridges.

At this time when the means of reproducing the natural tooth is so good, both as regards appearance and usefulness these operations are not frequently indicated especially when we remember that planted teeth only last from two to six years, and at best not more than fifteen years.

And at best the operation is quite severe on the patient even when an anesthetic is employed.

CHAPTER XVIII.

Diseases of the Soft Tissues of the Mouth.

Stomatitis. Aphthous Stomatitis. Treatment. Ulcerative Stomatitis. Mercurial Stomatitis (Ptyalism). Symptoms. Treatment. Eczema of the Tongue. Leukoplakia. Causes. Treatment.

Stomatitis.

The word stomatitis is of Greek origin and signifies inflammation of the mouth.

Acute stomatitis is the most common form of mouth inflammation, and is usually the result of some local or constitutional irritant. It is frequent in all ages and is often associated with acute attacks of indigestion following acute specific fevers.

In poorly nourished children it is often associated with dentition or may appear as a result of some gastro-intestinal disturbance. Tobacco users often suffer from this affliction, but more particularly have I seen it in the mouths of excessive smokers of cigarettes.

The chief characteristic of this disease is a general redness and dryness of the gums, sometimes extending to all the membranes of the mouth and lips, and the tongue may be slightly swollen, furred and indented along its margins by the teeth. All the mucous membrane may become tender to the touch and severely painful in mastication and particularly if hot drinks be taken.

Sometimes in children the temperature will rise one or two degrees. Treatment consists in removing the cause as far as possible and applying weak solution of nitrate of silver or chloride of zinc, three grains to the ounce, to the afflicted parts, and the frequent use of a borax mouthwash.

Aphthous Stomatitis.

This form is often spoken of as follicular or vesicular stomatitis. It usually appears in the form of small, slightly elevated spots about two mm. in diameter surrounded by a somewhat reddened zone; these vesicles soon rupture, forming ulcers with grayish bases and bright red margins.

The ulcer seems to sink into the tissue and the margins stand up above the surrounding parts. They most frequently occur on the margins of the tongue, inner surface of the lip and cheek.

They are usually associated with an attack of nervous indigestion, but many observers seem to be of the opinion that they may exist, as an independent affection. They occur in young children's mouths very fre-

quently, and I have served patients past the age of forty that suffer almost continually from them. The rapidity with which they form is a peculiarity of them.

The ulcers are usually very painful to the touch, making the taking of food difficult, and greatly interfering with dental operations, when present on the lips or cheeks. Young children complain of the pain and refuse to take food.

Treatment.

The local condition can usually be benefitted and the painful ulcer entirely relieved by touching it with a saturate solution of nitrate of silver. This remedy is so violently escharotic that care must be taken not to get it anywhere but on the ulcer, which will soon present a white coagulated appearance.

The ulcer should be kept dry for a few minutes after the application. One painting is usually all that is needed. An alkaline antiseptic wash should be prescribed, and patient referred to a physician for constitutional treatment if the local conditions are at all persistent.

Parasitic stomatitis or thrush, as it is commonly called, is a peculiar inflammation of the mucous membranes of the mouth. It is most commonly seen in very young children, and is dependent upon a peculiar fungus called the saccharomyces Albicans. The fungus belongs to the yeast family and has branching filaments. (Fig. 78.)

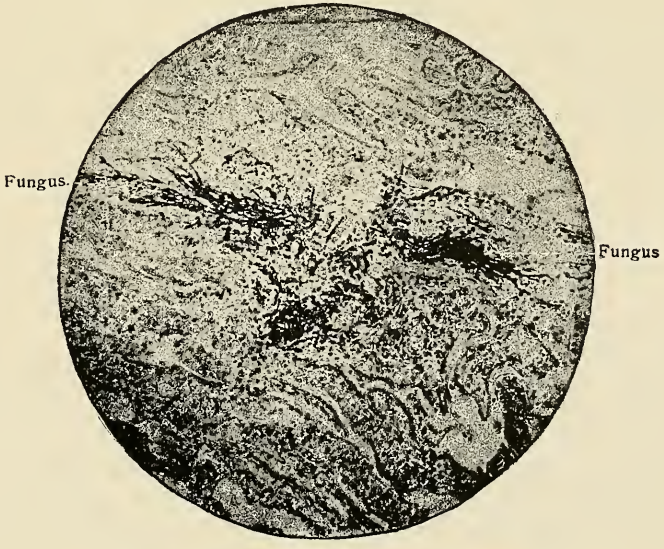

Fig. 78.
Ordium Albicans (thrush fungus). (Marshall.)

Osler says this disease does not arise in a normal mucosa, but may rapidly develop in unclean mouths, especially when catarrhal conditions of the mucous membranes are present, and acid fermentations of food remnants is permitted to occur.

This disease is not confined to children, but is often met with in adults during final stages of fever, chronic tuberculosis and kidney diseases. The disease usually attacks the tongue first, and then rapidly spreads to all mucous surfaces, and appears as slightly raised, pearly white spots which gradually enlarge and coalesce.

The parasite develops in the upper layers of the mucous membrane, and its filaments rapidly penetrate the epithelial cells, forming a dense network which can readily be scraped off, or if left alone in a short time will slough off, leaving a raw, bleeding, ulcerating surface, differing entirely from mucous patches which are confined to a small depressed area, while in thrush, the whole surface soon becomes involved.

I have seen a few cases where the entire mucous membrane on the lips, tongue, palate and cheeks were covered with what appeared to be a fuzzy grayish white membrane. A few cases are reported where this disease has extended to the soft palate, the throat and the stomach.

It is not often that the dentist is called upon to treat these cases, but he should be familiar with its clinical appearance.

It is epidemic in the spring and fall and in babies it is often the direct result of improper care of nursing bottles and nipples.

Treatment.—The child's mouth must be kept scrupulously clean and an alkaline wash used to relieve the burning sensation. A little lime water administered will often correct the acid mucous secretions and do away with the pain.

Osler suggests the use of a borax spray; the phenate of soda is also recommended.

Sudduth suggests the following prescription:

Acidi Carbolici	ʒ h i
Olei Gaultheriæ	℥ h ii
Olei Menthæ Peperitae	℥ h iii

M. Sig. Use as a spray.

In every case the patient's general health must be looked after by the physician, for in these cases it will usually be found that the child's nutrition is poor, presenting some marked disturbance of digestion.

Gangrenous stomatitis (concrum oris noma) is an affection characterized by a rapidly progressing grangrene usually beginning in the gums, and soon extending to the cheek, rapidly leading to sloughing and destruction.

It is the most formidable disease of childhood and fortunately it is very rare, and is seen only in children living under most unsanitary conditions or many times during convalescence from severe acute fevers. It is most frequently seen between the ages of one to five years, and is more common in girls than boys.

Some authors think it due to some specific organism, but it seems more like a coagulative necrosis. It usually attacks the gum high up on the buccal side of upper molar, and when first seen the mucous membrane only is affected but soon leads to induration of all the adjacent tissues; the sloughing extends and the blood vessels supplying the part are soon affected and the whole tissue of the cheek becomes hard and indurated and filled with pus and fibrin, and soon the cheek is perforated and intense inflammation spreads rapidly, and the tissues rapidly ulcerate away.

Severe constitutional disturbances ensue, the pulse rapid, temperature high, prostration very extreme and death within a week usually from perforation of a large vessel and hemorrhage; the child passing into a comatose state passing away without pain.

The treatment of this disease is usually unsuccessful. The constitutional conditions must be met, nourishments and tonics given.

The diseased parts should be burned with the cautery and nitric acid applied to the edges of the ulcers, and deodorant antiseptic lotions applied to destroy the very fetid odor which is always present.

When the disease is recognized and treatment begun early recovery may be hoped for.

Ulcerative Stomatitis.

This form of stomatitis usually begins at the free margins of the gums, which become red and swollen and bleed on the slightest touch. At the outset the mouth is hot and painful, and saliva flows freely, and the breath is offensive. The glands are usually swollen, and also the lips; a rash often appears resembling measles.

As the disease progresses ulcers appear along the gum margins of both upper and lower jaws. At the base of these ulcers is a firmly adherent grayish white membrane; and in very severe cases the edge of the alveolus may become necrosed and the teeth loosened.

It is a disease that rarely proves fatal, although death occasionally results in very debilitated children. The local treatment is to clean the teeth and treat the ulcers with dilute chloride of zinc, or what is better, the powdered chlorate of potassium applied directly.

Osler recommends the administration of chlorate of potassium in 10 grain doses, three times daily. Fresh air, wholesome food, proper elimination, are the essential requirements as general treatment for this, and all other forms of ulcerations about the mouth.

Mercurial Stomatitis (Ptyalism).

This is an inflammation of the mouth and salivary glands caused by mercury. It occurs in individuals whose business is associated with the constant handling of mercury and only rarely now as the result of mercury administration.

Twenty years ago this disease was very common when large mercury dosing was frequent and even now in an especially susceptible case mild attacks are seen, especially in individuals undergoing mercury treatment for specific disorders.

It should be said here that individuals differ greatly in this regard, and the susceptible cases cannot be distinguished beforehand, so that the physician needs to be on his guard and watch for the first signs of stomatitis, when the drug should be suspended for a time.

As soon as the gums are touched, the drug should be discontinued. To produce mercurial ptyalism it is not necessary that large doses be given, indeed many cases are recorded where a dozen doses of calomel 1-10 grain four hours apart produced serious results.

Symptoms.

The first indication of this affliction is a metallic taste which is soon followed by swelling and redness of the gum margins, teeth become sore and mastication difficult. The saliva begins to flow freely, and by and by runs out of the mouth. The breath becomes foul, tongue swollen, and after a few days ulceration of the gum margins appear and necrosis of particles of the alveolar border.

The teeth become loose and in many instances are lost or so affected that marked recession of the gum and absorption of alveolus results, which so weakens the structures that eventually the teeth are lost, and in a few instances necrosis of the lower jaw results. Marshall reports only two cases, both of which proved fatal.

Treatment.

The treatment must be largely symptomatic. The drug should be discontinued immediately or if the affection has resulted from the fumes while handling mercury the patient should get away from those surroundings. In either case the mercury should be driven from the system by the administration of alkaline waters in large quantities and frequent hot baths given.

If there is much salivation and accompanying prostration, the patient should be given atropine and other supporting treatment, liquid nourishment should be freely given, and if the pain is severe, Dovers powders should be given.

The local treatment consists in cleaning the teeth and gums as well as possible, and an application of chlorate of potassium mouth wash. If ulceration appears astringent mildly escharotic agents should be applied.

The case should be watched for several days, and if looseness of the teeth appears the application of iodide of zinc will be helpful.

A word should be said here regarding the general catarrhal inflammation of the membranes of the mouth, throat, and nose, which is so common in this climate.

In these cases all the membranes are more or less affected and present a decidedly reddened appearance more severe in some spots than others, and are usually accompanied by slight digestive disturbances. Unless some attention is paid to it more severe forms may result.

The treatment consists in correcting the digestive disturbances; and spraying the mouth, throat and nose with a carbolic acid and menthol solution several times a day until relieved.

Phosphorous necrosis.—This subject does not properly come under the scope of this book, and is only presented because of its similarity to mercurial stomatitis.

Phosphorous necrosis is the result of poisoning from fumes of phosphorous and is only seen in the individuals who work in match factories, fertilizer factories and the like.

Etiology.—There is some difference of opinion as to the exact mode of attack, but the preponderance of evidence seems to prove that the phosphorous necrosis is the result of local poisoning produced by the fumes entering the tissues through some break in the continuity of the mouth structures whereby they get access to the periosteum (Marshall), and carious teeth seem to be the most frequent route through which the fumes pass to the periosteum.

Symptoms.—This affection usually begins to manifest its presence by mild toothache and pain about the jaws; soon the pain becomes severe and swelling starts and spreads rapidly until the entire face and head are involved; it may affect one or both sides.

Abscesses rapidly form and may discharge on the face. The pus is very offensive and may exude around the necks of the teeth, which soon become loose, and small pieces of bone will often work out around the gum margin. Sometimes several teeth will be retained in a sequestrum of bone, which can readily be removed.

Treatment consists of supporting the general system, meeting any special conditions that may arise and surgically treating the necrosis.

Glossitis and eczema of the tongue.—The tongue is liable to the same forms of catarrhal inflammation as the other soft tissue of the mouth and

throat, and the treatment must be suited to each condition beside these local affections.

The tongue is the index to many disorders of the general system. In health it presents a moist, smooth, pink surface, and any change from this indicates some pathological conditions not usually of itself, but more frequently of the general system of which Sudduth gives the following interpretations.

"A white coated tongue denotes febrile disturbance; a brown moist tongue indigestion; a brown dry tongue, depression, blood-poisoning, typhoid fever; a red, moist tongue, feebleness, exhaustion; a red, dry tongue, inflammatory fever; a red glazed tongue, general fever; a tremulous, moist and flabby tongue, with blue appearance, tertiary syphilis.

"A moist, flabby tongue, with the imprint of the teeth in its sides, indicates general anemia; a pointed tongue shows intestinal derangement; a yellow furred tongue indicates bilious disorder; a moist tongue is a good indication of sickness; while a dry tongue represents the converse condition."

Glossitis signifies inflammation of the tongue. It may be either acute or chronic.

The acute form may often be the result of careless use of dental instruments. Recently I attended an alarming case where the tongue was badly lacerated as a result of tooth extraction. The patient had been put under gas anesthesia and accidentally the mouth prop slipped out of place and the jaws closed, and the tongue was caught between the teeth, resulting in cutting and tearing the tongue badly; infection resulted, followed by very alarming symptoms, several abscesses formed in the tissues, a rise of temperature to 104 degrees, and almost total collapse from the pain. The tongue was so badly swollen that deep punctures with a bistoury were necessary.

I have also seen a few cases where the tongue was infected from injuries made with broaches, scalers, forceps, etc.

Treatment.—In every case the conditions present must govern the treatment. The general elimination must be looked after, abscesses opened and drained, ulcers cauterized and general antisepsis maintained. The tongue usually makes quick recovery where proper treatment is instituted.

Eczema of the Tongue.

This is a disease of the tongue which is characterized by remarkable sloughing off of the outer epithelium. It usually begins in small patches which gradually spread, uniting with one another, presenting an irregular shaped raw surface which has been likened to a geographical map.

It is usually accompanied with a good deal of itching and burning pain. The etiology of the disease is not known and treatment consists in frequent applications of solution of nitrate of silver.

Leukoplakia.

Leukoplakia buccalis is a subject that has received considerable attention of late from many writers. The term leukoplakia is derived from the Greek and signifies white plates or plaques.

The affection is often referred to as buccal psoriasis, leucoma, ichthyosis lingualæ, smokers' patches and superficial glossitis.

Leukoplakia is a chronic inflammation of the mucous surface which manifests itself in irregular thin, smooth patches, white or pearly white color, which show no tendency to ulcerate. The spots are hard and resemble corns, indeed they are sometimes termed lingual corns.

The ichthyos variety, however, resembles warts more than corns and are slightly raised.

Location.—The plaques most frequently appear on the dorsum of the tongue, but many may be seen on its margins and all the mucous surfaces of the mouth.

Marshall reports several cases where the beginning was on the gingivus, others on edentulous spaces in the upper jaw, and a few on the buccal gum of the lower jaw.

Etiology.—The etiology of leukoplakia is not well understood and marked difference of opinion is expressed by different writers. Some writers regard it as a mouth manifestation of psoriasis and others think it a form of skin disease resembling herpes zoster or hives.

Many seem to regard it as due to syphilis, and still others think it is a purely local affection induced by smoking tobacco. The bulk of evidence seems to point out that this is a distinct affection, and while it resembles some mouth manifestations of other diseases still it does not often appear in connection with any of them.

Women seem to be almost entirely free from it so far as I can learn, and it rarely manifests itself in men under twenty-five and most frequently about the age of fifty. A little experience in observation of these cases will enable the dentist to distinguish these plaques from syphilitic mucous patches.

The latter always present a curdy grayish white appearance and are slightly raised above the surrounding tissue. Syphilitic patches show a tendency to ulcerate and discharge a thin watery fluid and on the surface look like a corroded spot and always yield readily to treatment.

If any doubt exists a short course of mercury treatment will clear the matter up. The dentist should be familiar with all these mouth affections.

Leukoplakia plaques may extend in size and become papillomatous and many instances are recorded where genuine epithelioma was developed from them.

Marshall calls special attention to this affection for following reasons. 1. It is an exceedingly dangerous affection, often being a forerunner of carcinoma. 2. It is a disease which from its innocent appearance and the painless character of its early stage is seldom recognized until the disease has progressed to a stage which renders a favorable prognosis exceedingly doubtful. 3. The disease seems from personal observation to be on the increase. 4. The dental surgeon, from the very nature of his specialty, is in a position to see and recognize the disease in its earliest stages, and to warn the patient of his condition before it has progressed so far as to prove a menace to life.

The disease in its earliest stages is much more likely to come under the notice of the observing dentist, or stomatologist, than of the surgeon or the laryngologist. As a rule the patient does not consult a surgeon until the disease becomes troublesome; it may then have progressed so far as to give unmistakable evidences of degenerative changes of a malignant character.

The dentist therefore should be so familiar with the characteristic features of the disease that he could recognize it at a glance; while it would be his duty to impress upon the patient the urgent necessity of consulting an oral specialist with the view of instituting measures calculated to arrest its further development, or for its complete extirpation.

Causes.

The causes of leukoplakia seem not to be well understood. Many writers seem to think there is always a peculiar thinness of the mucous membranes prone to eruption of every sort and regarded as especially delicate. Many claim that chronic dyspepsia and gout predispose the individual to this affliction.

The exciting cause is charged to the use of tobacco, particularly the pipe and cigarettes, and the use of undiluted spirituous liquors; but any constant irritant such as ill-fitting plates, rough teeth, large amount of salivary calculus may be exciting causes also.

Treatment.

This affliction does not readily yield to treatment, and most authors recommend that it be let alone, but a few suggest the use of a 1 per cent solution of chromic acid applied directly to the patch.

All sources of irritation should be removed. When the spots take on a papillomatous form they should be surgically removed. Many authors recommend this procedure for all forms of the disease as the only means of cure.

CHAPTER XIX.

Oral Manifestations of Syphilis. General Considerations.

Location. Source of Infection. Diagnosis. The Dry Scaling Papule. Pathology. Positive Diagnosis. The Secondary Stages of Syphilis. Secondary Eruption. Treatment. Tertiary Syphilis. Congenital Syphilis. An After Word.

At the outset I wish to say that no description that I can give will convey such accurate knowledge of the appearance of primary syphilis as can be obtained by seeing a few cases.

"Syphilis is a general infectious disorder, both acquired and transmissible by inheritance, chronic in course, and displaying in a determinate order specific symptoms."—Hyde. It is produced by a specific microorganism whose identity has not been definitely established.

The history of the disease extends as far back as we have any records, and its ravages at the present time are simply appalling. "The infection is always conveyed from one to another either by direct contact or through the medium of some instrument, utensil or other article upon which it has been deposited."—Baldwin.

Before infection can occur it is not only necessary that the virus be present, but it must gain entrance to the system through some abraded surface. It cannot enter through the healthy skin or mucous membrane, but so virulent is this disease that if the virus does find such an entrance to the circulation syphilis will certainly develop.

Location.

Next to the genitals the mouth is the most frequent location for the primary lesion, and it is because of this fact that dentists should be very familiar with its many manifestations. It has been stated by several syphilologists that over 70 per cent of all extra genital chancres are found in the mouth, tongue, gums, tonsils, avula and buccal mucous membrane, and of all these, the lips and tongue are most frequently affected.

The most frequent modes by which transmission of virus occurs are kissing, passing around smoking pipes, using common towels and handkerchiefs, drinking vessels, knives and forks, blow pipes, whistles, wind-instruments, tooth brushes, and, in the case of infants, the nursing bottle is a frequent source.

Source of Infection.

The infectious material may come from both primary and secondary lesions and from the blood of the victim. These secretions when deposited

on towels, etc., and allowed to dry are still infectious during these periods, but all the other secretions of the body are regarded as non-infectious, as is also true of all secretions in the tertiary stage.

Diagnosis.

When infection occurs there is within twenty-one days a primary sore developed which is termed a chancre. The period of incubation varies from six to thirty days. These chancres are always single and are not auto-inoculable. They differ in appearance, each presenting certain marked characteristics.

These variations may depend on accidents attending inoculation, peculiarities of the individual, or on the particular tissue affected. The typical and most usual form of chancres as seen on the lip is what is known as the Hunterion or ulcerating chancre (Fig. 79).

Fig. 79.
Chancre of the upper lip. (Barrett.)

In common with most forms of chancre, this variety begins in a small papilla, which very soon sloughs off its apex and presents a deep red round or oval ulcer. The margins are usually elevated and very red,

while the center is concave and presents an ulcerating appearance and secretes a thin serous fluid, and not infrequently this sore may become infected with pus or other germs, when a mixed infection occurs, resulting in deceiving complications. This form is always on the mucous surface.

Superficial chancrous erosion is a form frequently met with in the mouth. It begins with a little round or oval red spot, from which the epithelium soon sloughs off, giving the appearance of a raw sore, which is never deep and presents a smooth surface, which occasionally may be covered with a gray film, which makes it easily mistaken for leukoplakia or aphthous stomatitis.

The duration of this form of chancre is usually short and often goes unnoticed until constitutional symptoms appear.

The Dry Scaling Papule.

This variety begins in a small dull red slightly elevated papule, which gradually elevates and turns purple; it is hard to the touch. The outer epithelium is soon lost, presenting a dry crust. When found in the mouth is usually seen at the junction of two mucous surfaces, and those I have seen were high up on the buccal gum over the upper third molar.

The incrusted chancre is the variety usually seen on the skin, and is the form usually seen when the finger or other part of the hand has been accidentally infected.

The indurated nodule is the form usually seen at the junction of the skin and mucous membrane, particularly of the lip. It has a sharply defined plaque, or nodule, elevated with sloping edges and is dry.

When the primary sore appears on the lip, it is most apt to occur in the fissure in the middle of the lower lip or on the upper a little to one side of the median line (see Fig. 79). Of course it may occur at any other abraded point.

As previously stated, it is the ulcerating chancre that usually appears on the lip and presents a vermilion border with red or grayish base. When it occurs on the outside of the lip it usually presents considerable induration, but when within the lip little or no induration. The submaxillary glands are always enlarged.

When the chancre occurs on the tongue it is usually flat or very slightly elevated, usually reddish, and when just at the tip the appearance is often as though a piece had been cut off. It is usually sharply defined, and can readily be distinguished from ordinary aphthous patches by its elevated border and less concave center (Fig. 86), but when doubt exists watch for the enlargement of the suprahyoid glands.

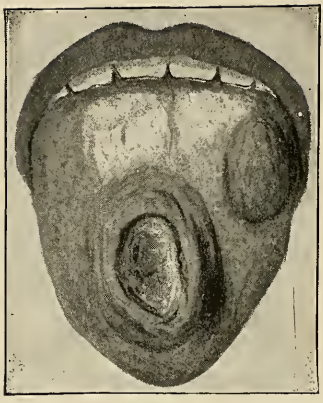

Fig. 80.
Syphilitic ulcer on the tongue. (Black.)

Pathology.

The pathology of the initial chancre is one of unusual interest. When the virus is planted an interesting cycle of phenomena at once begins. "Here if it finds favorable soil it grows and slowly increases till its intrusive presence becomes a source of offence to the tissues harboring it, and gradually inflammatory reaction sets in."—Bronson.

At first it does not appear that the virus passes directly into the circulation, or if it does it is in such small quantity that it does not make an impression, for not until after the chancre appears do we see any manifestations of general infection.

The pathological changes in the tissues at the point of infection, are described by Baldwin as follows:

"When the poison of syphilis is deposited on an abrasion in otherwise healthy skin, a cycle of phenomena at once begins. The first manifestation of this cycle is the infiltration of the tissue at the site of infection with small round cells, exactly as in any inflammation. With these small round cells are also to be seen large round or oval and polyhedral cells, filling up the interstices between the meshes of the network of blood capillaries.

"At first the blood vessels are involved, but by extension they are included in the inflammatory process. No connective tissue of a perishable or embryonic type is formed. This tendency to connective tissue formation is also observed in the tertiary stage in lesions of the nervous system due to syphilis.

"The lymphatic channels are soon involved in the inflammatory process, and the virus, which is either a microbe, or, as Otis suggests, a microbe-bearing cell, is borne along these vessels to the nearest lymphatic glands,

it is deposited, and the same process of inflammation is repeated and the glands become swollen and indurated. 'First intuition virus' of infection has traveled. This is the period of first incubation."

The microscopical changes are at first those that occur in all forms of local inflammation. The white blood cells flock to the seat of infection and are soon modified by the action of the virus, and it is the cells that carry the infection to other parts of the body, particularly to the lymphatic glands.

Chancroid is a soft chancre which does not incur any constitutional symptoms. It is of a pustular form and its secretions are infectious and also inoculable, and hence they are usually multiple. It is never seen in the mouth and causes no mouth lesions, and therefore has no special interest to dentists.

Positive Diagnosis.

I scarcely need remind the reader that it is not always possible to distinguish the beginning of chancre in the mouth from many other mouth lesions, and while I have tried to point out the distinguishing characteristics they are not always an infallible guide, and even in the more advanced stages it is rarely wise to trust the history obtained from the patient, for they will usually deny any knowledge of it, some because they do not know it and others because they wish to conceal it from the dentist; and yet every sore on the lips and in the mouth should not be suspected to be of specific origin, and we should be careful about alarming patients by stating positive conditions until they are positively proven.

Many of these cases are first seen by the dentist, for when the primary sore appears in the mouth the patient usually consults the dentist, thinking it to be associated with some affection of the teeth.

The first duty of the dentist is to obtain the history of the case, even though in some instances he may have to listen to a carefully concocted story as to how the infection occurred, that will more than tax the credulity of the novice.

To one who has a little experience these stories serve as convincing proof of the conditions present. If there can be no definite history obtained then we must wait the development of positive proof, which if not immediately seen in the characteristic appearance of the sore will be cleared up in a few days by the appearance of glandular enlargements and indurations, which ushers in the period of second incubation.

From quite an extensive experience in handling syphilitic cases in all stages of the disease, I find that nearly every sufferer is glad and anxious to furnish you all the information at his command.

The treatment of the primary lesion is to apply some form of cautery, usually nitric acid, nitrate of silver or chromic acid, but the most important thing is to clean the mouth and put the patient in the hands of a competent specialist, who will put him through a thorough course of treatment which will eventually drive the virus from the system.

This treatment consists in the use of mercury and potassium salts, which are pushed to the limit of tolerance, each alternating with the other. This disease is considered very amenable to treatment, but it requires from two to five years to accomplish a cure, which cannot even then be considered permanent or positive.

The Secondary Stages of Syphilis.

While the primary manifestations of syphilitic infection is in a local lesion or chancre, sooner or later distinct signs appear which show that the whole system is being infected. These signs, which are at first scarcely noticeable, become pronounced toward the end of what is known as the second incubation period.

"In distinction from all other known infectious diseases, syphilis has a second period of incubation, by which is meant the time elapsing between the advent of the so-called secondaries—a generalized eruption and its concomitants."—Ziesler.

The secondary manifestations of syphilis begin to appear about the fourth week, although it should be stated that sometimes they appear in twelve days, and occasionally they never appear, the patient skipping periods of several years, when the tertiary or third stage may appear.

The secondary manifestations are usually ushered in by a short period of fever and fatigue and headache, particularly over the frontal region. As soon as the initial sore has taken root there is some general morphological changes in the blood and the glands in the neighborhood become indurated, especially the small glands about the neck and behind the ears; they attain the size of small peas.

It is probable that the virus is carried throughout the system through both the lymph and blood vessels. The hair begins to fall.

Secondary Eruption.

The recognition of the secondary eruption is very important because it not only confirms the diagnosis, but its secretions are also infectious, and the mucous patches which usually develop are most frequently the source from which infection is disseminated.

These eruptions begin in the form of a rash known as syphilides, and which assume every variety of form that the nature of the skin and mucous membrane will permit.

Syphilides are always situated in the papillary layer of the skin, and appear at different parts of the body; they have their beginning in a kind of roseola or skin redness over the abdomen, front of the legs, back of the neck and scalp.

Fig. 81.
Syphilides on the face. (Fox.)

The face is not so often affected except in the more severe forms (Fig. 81). The mucous membranes of the mouth are often affected. These syphilides may present in any or all of the following forms: erythematous (red blotches), copper colored spots, scaly spots, vesicular pimples, tubercular nodules, rupial or crusty form; all of these forms may exist on the same subject and many intermediate forms as well. The first manifestation in the mouth of this secondary stage is the presence of erythematous spots all over the palate and fauces.

In the mouth the mascular copper colored spots are frequently seen; they occur on the tongue, hard and soft palate, mucous membrane of the cheek, and especially underneath the tongue in the mucous membrane folds.

The papular form begins as a reddish pimple on the skin, but in the mouth as an erosion and not as a pure type. They are situated in the submucous structures and present the appearance of a raw sore with sharply defined edges which are slightly raised, sinking in the center. The bottom may be red or slightly yellowish, the discharge is not great, but it is regarded by syphilologists as the most infectious of all the secondary mouth lesions.

The other variety of syphilides with which the dentist has most to do is known as mucous plaques or mucous patches. They are most frequently seen on the border of the tongue, the inside of the cheek or inner surface of either lip.

In general appearance they resemble the ordinary aphthous patches which are so frequently seen, and which have been spoken of in the preceding chapter. Syphilitic mucous patches assume two forms, one erosive, with a moist, brownish red surface, with decided hypertrophy around its margins; under the finger they are hard and are warty in appearance.

The other form present is milky white secreting center, which is somewhat depressed, surrounded by a red elevated margin; the secretions are foul smelling and infectious. The number of these patches present in the mouth of a given case depends somewhat on personal uncleanliness, decayed, rough teeth, using hot foods, alcoholic beverages and tobacco smoking.

Treatment.

The treatment of these mucous patches is very important, for, as stated, the secretion from them is very infectious. In addition to the proper mercury course each ulcer should be cauterized with nitrate of silver, or chromic acid 10 drch. to the ounce, and suitable germicidal mouth wash prescribed and all sources of irritation removed.

Tertiary Syphilis.

While the tertiary form of syphilis usually follows the secondary stages it does not always develop. Many cases are terminated by proper treatment and no tertiary symptoms appear.

The second and third stages may be merged into one and many authorities consider the third stage only the sequelæ of the other forms and not true syphilis; but be that as it may, the fact is that this form usually follows the other, and is, strictly speaking, the destructive stage.

This stage is usually manifested by more or less rapid destruction of both hard and soft tissue, and is, strictly speaking, an ulcerative process, in which the tendency is to eat deeply into the tissue and spread in all directions. Fig. 82 represents a case in which a hole was eaten through the hard palate.

This stage usually begins with the appearance of tubercles or superficial gumma, which appears in skin and mucous surfaces, and as time goes on the deeper structures are involved and the surface begins to eat away.

Gummata are often seen in the mouth, particularly on the tongue (Fig. 83). They usually begin as a collection of small round spots, which

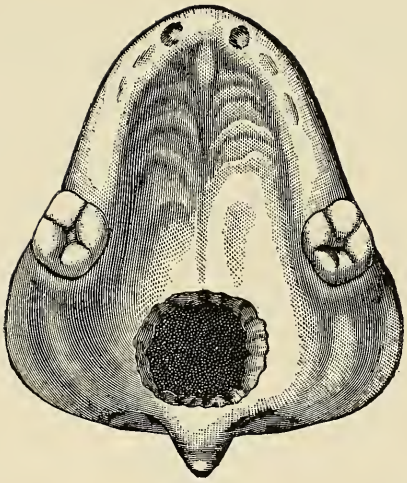

Fig. 82.
Syphilitic ulceration of hard and soft palate. (Marshall.)

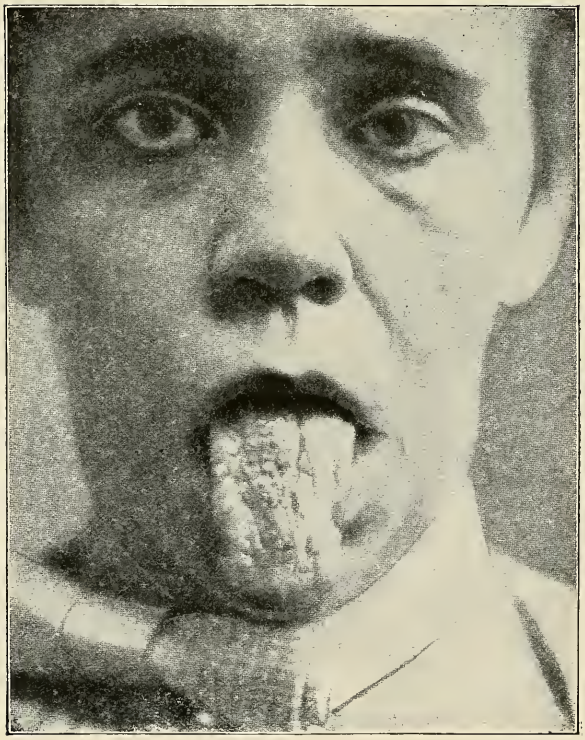

Fig. 83.
Gumma—toad's back appearance of tongue in syphilis. (Wende.)

seem to pain and soften, and finally break down and begin their peculiar process of ulceration, which gradually spreads to all the adjacent tissues.

The margins of these ulcers are usually irregular, overhanging, and often small pieces of soft tissue will be cut off and float away. The secretions from these ulcerating surfaces, while exceedingly foul smelling, is not infectious.

The dentist is not called upon to treat the latter stages of this affection, and yet a knowledge that will enable him to recognize it at sight is important, for during this stage no serious operation should be undertaken, especially if it contemplates the cutting or lacerating of tissue to any extent; even the extraction of a tooth often leaves a wound that is very slow to heal.

The temptation to cut the gummata is very great unless one recognizes their character.

Congenital Syphilis.

It is a lamentable fact that this disease is hereditary and that so many infants are born into the world with a syphilitic taint.

The disease may be transmitted to the offspring by either father or mother, and the father may transmit it without infecting the mother. Many times the disease proves fatal to the fetus, and more often the child dies soon after birth; but there are many cases where the taint is not shown until considerably later in life.

It always begins in the tertiary form, and is therefore not infectious, and in this connection it should also be said that it is very much more resistant to treatment than when acquired.

It usually manifests its presence by a peculiar erythematous rash, although often at birth there may be other unmistakable signs such as cracks in the lips and fissures in the tongue, rough nails, cleft palates and other indications of faulty development.

Hutchinson's teeth are no longer considered signs of syphilis, but rather signs of some disturbance of nutrition during the period of tooth formation. If none of these signs appear during the first year the anxious parents may feel quite certain that their child has escaped the taint.

An After Word.

Syphilologists have discovered that they can push the mercury treatment safely and to a much farther extent without danger of salivation and necrosis; when the mouth is in a healthy condition, so that it has become a custom among them to first send these syphilitic cases to the dentist that his mouth may be put in order, particularly relating to thorough cleansing of teeth and gums.

Many of these cases apply to the dentist before they do to the physician because, as stated before, they often think the initial mouth

lesion due to some disorder associated with the teeth; the dentist, therefore, should be able to recognize the condition, and after putting the mouth in order as to cleanliness, the patient should be directed to a competent specialist.

In this regard it should be stated that only temporary work in the way of fillings is attempted until the proper course of treatment has been carried through by the physician.

Crowns and bridges and all work of that nature is not attempted. Dentists must use every precaution in handling these cases, both in regard to avoiding infecting themselves through sores, scratches, hangnails or any kind of abrasion on the hands; and when cleaning teeth avoid specks of tartar or débris flying into the eye.

The care of instruments is spoken of in Chapter XIII, to which the reader is referred. I ask the reader to familiarize himself with this disease; learn to recognize it at a glance by observing well understood cases, many of which can be found in almost every hamlet.

That the dental profession is sadly ignorant of the manifestations of this disease cannot be denied.

That many operators work day in and day out, utterly careless of the dangers of carrying this virus from one mouth to other mouths, or of infecting themselves, is equally true.

From the facts I have tried to present regarding this disease it must appear to be of very serious importance to the dental profession, for how culpable is the operator who unwittingly or even through ignorance of its nature infects an innocent human being with this most awful malady, a disease which, though now considered amenable to treatment, always leaves in its wake not only death, but living destruction, shame, loathsomeness, rottenness, paralysis, and horrible markings to untold thousands of the best families of our country.

CHAPTER XX.

Diseases of the Maxillary Sinus.

Empyema. Etiology. Symptoms and Diagnosis. Treatment. Ulcers. Necrosis. Causes. Treatment. Tumors.

The maxillary sinus, or antrum of Highmore, is a triangular shaped cavity contained within the body of the superior maxillary bone. It has a natural opening into the nose through which the normal secretions pass, and is lined with a mucous lining which is analogous to the schneiderian membrane of the nose.

This membrane is covered with ciliated cells so arranged that in normal action they carry the secretions out through the opening into the nose. This opening is not at the most dependent part of the antrum, and hence this provision of nature.

The floor of the antrum is immediately over the apices of the roots of the posterior teeth, and in many specimens examined the floor is convoluted, each little eminence being over a root apex.

Antrums differ both as to size and shape; in some cases its anterior wall is as far forward as the cuspid root apex; in others it does not come forward to the second bicuspid.

Most antrums are divided into two or more chambers by thin bony partitions arising from the floor to about one-quarter the height of the sinus, and many cases have no divisions. The antrum serves as a sounding board for the voice.

Diseases of the antrum are very common, more so than formerly, especially among the lower classes; and climatic conditions have much to do with its prevalence in certain localities, and certain constitutional conditions have much to do with these diseases.

The diseases of the antrum can be divided into four general classes.

First—Empyema, a suppurative inflammation.

Second—Ulcerations of its mucous lining.

Third—Necrosis of some portion of its walls.

Fourth—Tumors. The last two requiring essentially surgical treatment.

Empyema.

Empyema of the antrum is a purulent inflammation affecting the antrum.

Etiology.

Empyema often has its source in acute or chronic catarrhal inflammation, which may come from the nasal cavity on account of its close proximity. Inflammation of the membranes of the nose may result in partial or complete closure of the natural antral opening, and as a consequence stagnation of its fluid contents, which in time may become infected with pyogenic germs, resulting in suppurating inflammation, with breaking down of the lining membrane. This is probably the most common cause.

Another point that should be mentioned here which may act as a causative factor, and that relates to the fact that sometimes the secretions of the frontal sinus and the ethmoid cells, instead of discharging through the infundibulum into the middle meatus of the nose, discharge directly into the antrum. This fact was pointed out by Cryer.

Abscessed teeth are a frequent cause by discharging pus into the antrum. The presence of foreign substances such as roots of teeth that have accidentally been forced there in extracting. Malposed teeth have been found erupting into the antrum.

Symptoms and Diagnosis.

The symptoms of this form of antrum disease are sometimes misleading, particularly if the case is one of chronic slow suppuration, when the discharge will often not be noticed. In the great majority of cases there will be an offensive odor, a feeling of fullness, a discharge into the nose when lying on the opposite side of the face, or in very acute cases, where suppuration is rapid and opening partially closed, there will be severe pain and swelling, pain often in the eye, and occasionally pus discharging into the mouth through a sinus in alveolus where tooth had recently been removed.

Examination through the nose by use of nasal speculum and probing needle pus or other purulent fluids can be extracted. Trans-illumination will sometimes assist.

Treatment.

The treatment of the usual engorgements of the antrum where there is no pus or local lesion is very simple. It consists in reopening the natural opening into the nose and expanding it with a trocar or inserting a piece of tubing to allow free drainage and douching with warm normal salt solutions. A few treatments will usually suffice if the nasal conditions are attended to.

In severe suppurating cases it is always best to secure an artificial opening; it is best to make this opening into the mouth because it can be made in the most dependent part of the sinus, and is more easy of access

and consequently better drainage. I have never had any success in treating these cases through the nasal cavity, although some recommend that method.

If the first molar is missing it is easiest to make the opening through its socket. If all the teeth are present then I like Dr. Gilmer's plan of opening above the mesio-buccal root of the first molar, where the cheek will close it and keep food out.

A small opening is all that is needed at first, although it must be large enough to furnish ready drainage. The antrum should then be irrigated with copious quantities of normal salt solution, repeating every day for a week, when recovery will result in the simple cases; but if considerable foul smelling pus be present then more radical measures are necessary.

In these bad cases I usually proceed as above for a treatment or two, then if pus continues, I make a large enough opening to place the small electric bulb inside the antrum to light it up, and not only explore with an instrument but with the finger.

Sometimes the bony partitions may have to be broken down, and of course if there is any dead necrotic tissue it should be removed and the lining carefully curetted, and all flushed with normal salt solutions and borax water with cinnamon and carbolic acid. After irrigating freely the sinus should be packed with iodoform gauze.

Irrigation and repacking should be repeated every few days according to conditions until all pus ceases. In cases of violent pus formation, I use 1 per cent chinosol solution as a final douche and pack chinosol gauze a time or two.

In addition to this the patient should use solution of Siler's antiseptic nasal tablets with which to douche the nose twice daily. It is sometimes advisable to use a gutta-percha plug to keep the opening from closing. This can usually be kept in place by clasping it to an adjoining tooth.

Ulcers.

Ulcerations are usually the result of some constitutional disturbance and usually affect the mucous lining of the nose and mouth. The only ulcerations of serious moment are associated with syphilis, which sometimes destroys not only the linings but periosteum and bone.

The treatment must be constitutional, using those remedies indicated for tertiary syphilis. The local treatment consists in keeping the parts clean.

It is sometimes difficult to make these artificial openings close perfectly. In several cases I have scarified the edges and sutured together.

Necrosis.

Necrosis of any of the walls of the antrum is a possibility, but I have never seen any except those involving the floor and outer wall.

Causes.

Necrosis may result from the same variety of causes as that occurring in the other portion of the body, but most commonly from inflammation of the periosteum as a result of some disease associated with the teeth or traumatic injuries.

Alveolar abscess may result in alveolar necrosis involving the floor of the antrum, and the outer wall may be destroyed as a result of pus poisons in the antral cavity.

Treatment.

In either case if a sequestrum has formed the necrotic bone should be surgically removed and treatment instituted the same as for empyema. There is one additional point which relates to holding the contour of the face after outer wall is destroyed. This is usually done with antiseptic wax or gauze packing until such time as nature can supply the needed bone.

Tumors.

Tumors of the antrum may occur in every variety, but most commonly as polypi and other mucous cysts. They are usually very vascular, usually arising from the floor, and rarely attain such size as to cause serious trouble, although some authorities claim they are malignant in their tendencies. The treatment is a radical surgical one, in which a generous opening into the antrum must be made either through the nasal or outer wall, through which the tumor can be thoroughly removed, and after treatment similar to that already described.

CHAPTER XXI.

Management of the Diseases of Children's Teeth.

Dentition. Pathology. Treatment. The Diseases of Deciduous Teeth, and Soft Tissues of the Mouth. Diseases of the Pulp. Putrescent Cases. Root Filling. Sensitive Dentine. Cleaning Teeth. Management of Permanent Teeth During Childhood. Management of Sensitive Cases.

The pathology and therapeutics of the diseases common to children's teeth can most easily be presented under three heads.

1. Those diseases incident to the process of teething.

2. The diseases of the deciduous teeth and soft tissue before the permanent set are erupted.

3. The care of permanent teeth during childhood.

Dentition.

Dentition may be defined as the process of teething; it is the physiological process of supplying the infant with teeth. The process may be said to begin when the crown of the tooth has formed and begins to pass through the bony covering in which it is held.

It must be remembered that the early calcifying tooth is contained in a bony crypt which is separated from the bone of the jaw by vascular tissue on all sides. In the lower jaw the floor of the crypt rests immediately over the inferior dental canal, and in the upper jaw it rests over the infraorbital canal.

The covering of the crypt is a thin layer of bone which forms the outline of the alveolus. It can readily be seen then that the roots cannot form until the crown passes toward the surface away from the canals in the jaw.

The bone over the crypt is slightly fissured to facilitate absorption and make the passing through of the tooth crown easier. As the crown passes through this bone the roots begin to develop and continue until some little time after the tooth crown has assumed its position in the mouth.

While this is going on there is gradually forming an alveolar wall which is to make the tooth socket. This is the method by which all the teeth develop and take their places in the arch, both upper and lower.

It does not seem to come within the province of this article to present data regarding the time of eruption and calcification of the various

teeth, which can be learned by referring to books on dental anatomy. What we are interested in now is the diseases associated with the process of dentition.

While the process of tooth erupting is physiological it is nearly always associated with disturbances which are pathological. The process by which the tissues over the erupting tooth are forced out of the way is one of resorption under pressure.

The teeth cusps act as the irritant which produces the stimulus to the resorption process, and consequently the tissues must in themselves be tender and a source of considerable pain.

A glance at the tissues will reveal the hyperemic condition present, which as the tooth presses through the gum tissues often becomes inflammatory. The pressure on the nerve filaments must be a source of considerable pain. The parts become hot, which is shown by the infant's desire to bite on something cold.

It is doubtless true that the desire to bite things is an effort of the child to relieve the pressure irritation, and yet it seems to be a provision of Nature by which the gum is forced out of the way of the erupting teeth, and hence the value of the ivory or silver ring, which can be kept clean, and affords a means by which the infant may aid nature.

So long as the process does not exceed the bounds of a reasonable physiological process very little disturbance results; but when either from the density of the tissues or other complicating circumstances such as faulty nutrition the normal process is interfered with, then we often have a train of consequences which may even seriously endanger the life of the infant.

When we remember that the nerve which supplies these tissues is the fifth cranial nerve, and that this is the largest and most sensitive of all the nerves, and that these tissues are so intimately associated with the great sympathetic system, we can readily see why such grave disturbances occur.

Coupled with this, also, is the fact that at this period of life the spinal system predominates the system. Then, again, the mucous lining of the mouth and tongue are in such close proximity to the throat, œsophagus and stomach, that the affection of the mouth can readily spread to these organs, which are necessarily sensitive to environment.

When we take all of these things into consideration we can see a rational explanation why sometimes such serious disturbance may be rightly attributed to faulty dentition. However, I am quite convinced that much mischief is laid at the door of erupting teeth which does not rightly belong there, but which for lack of better understanding of the

real cause can easily be explained to the satisfaction of mothers by saying, "your baby is teething."

Pathology.

The first indication of teething is seen in the increased flow of saliva. This "drooling" is due to irritation of the fifth nerve, which in turn affects the salivary glands through another of its branches. This is evidently a plan of nature to cool and keep moist and clean the parts.

The next indication is the cheek eruptions, which are doubtless reflex in origin. Sometimes this takes the form of mucous ulcers, which are sore and must cause a degree of pain and restlessness. The child usually becomes wakeful and peevish, and if the gums become severely inflamed cries and displays "fits of temper." If several delayed teeth are erupting at the same time and the consequent local condition unusually severe, diarrhœa, colic and even convulsions may develop.

Miller points out that the germs which cause infant diarrhœa are usually found in the mouth, by which route they probably enter. I should add here that frequently carelessness about sterilizing nursing bottles and nipples is responsible for many serious mouth affections.

In examining the mouth of an infant in such distress the thing to look for is evidence of severe active inflammation over the region where, according to the age, the tooth should erupt, and then such other sore places as can be found.

Treatment.

If severe localized inflammation presents then the thing needed is the lancet used under antiseptic precautions. It is not so much to remove the gum over the tooth (although it is as well to cut deep) as to let the congested blood out that the lancet is used. The accompanying illustration will indicate the best method of lancing (Fig. 84).

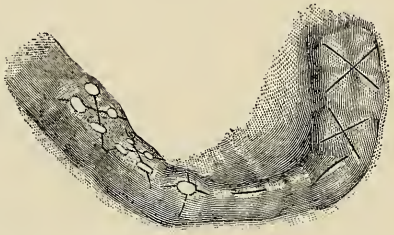

Fig. 84.
Showing method of lancing gums over erupting teeth. (Burchard.)

Care must be taken to hold the infant securely and guide the lancet so as to avoid all danger of slipping and doing serious damage to adjacent parts.

The parts should be carefully sponged with a boric acid solution and the constitutional disturbance attended to by the physician.

The Diseases of Deciduous Teeth, and Soft Tissues of the Mouth.

The care that the temporary teeth receive has much to do with the value of the permanent set, both in relation to their formation and position in the arches. The child should be brought to the dentist early in order that its teeth may be examined and whatever treatment necessary given, and wholesome instruction given both parent or governess and the child.

The objects of caring for the deciduous teeth are three: First, that they may be preserved to do necessary mastication until permanent teeth erupt; second, that the normal process of development may prepare the way for normal occlusion of the permanent set, and third, that the child may avoid a series of painful conditions which will result in severe operations and consequent everlasting dread of the dental chair.

The dentist should do all he can to correct the false notion in the minds of parents that these teeth are unimportant because temporary.

There is another point in this connection and that relates to the cultivation of good hygienic mouth habits, which once established will continue through life, and also the development of that friendship between dentist and child which will entirely do away with that awful dread of the dentist and dental operations which so many experience.

Diseases of the Pulp.

Temporary teeth are subject to all the diseases common to the permanent, but in a modified form.

Hyperemia of the pulp results from the same causes, but it rarely produces such severe pain. This is accounted for by the fact that the canal has usually begun to enlarge by resorption before such conditions arise and also the lymphatic connections are better.

The treatment must always be palliative. The carious cavity should be cleansed as well as possible of all loose material and anything pressing on the pulpal wall removed, after which the cavity should be dried and an application of iodoform and hydronaphthol made into a paste with pure oil of cloves should be made, when the cavity can safely be filled with cement, gutta-percha or amalgam, as seems best suited.

Unless positive infective inflammation has begun such cases will usually get well and remain so. If, however, infective inflammation has begun, which can usually be determined by the history of the case and the presence of an exposure, then the procedure must be different.

The destruction of the pulp in temporary teeth should not be attempted with the use of arsenic; the chances of it passing through the

wide open root and affecting the underlying tissues is very great, as well as the danger to the surrounding parts.

Many operators suggest the use of corrosive agents such as zinc, chloride, silver nitrate or carbolic acid, which will often work well; but I find the ordinary anodyne treatment, with the use of clove oil and the like, to serve fully as well, which will keep the tooth quiet until death of the pulp results, when it can be cared for. It should be said, however, that exposed pulps of deciduous teeth take more kindly to capping than do the pulps of permanent ones.

Putrescent Cases.

In the management of putrescent and abscessed cases the same general principles obtain as in permanent teeth with this exception, that corrosive agents are never needed.

The important points are to mechanically clean the canals, and force some oil of cloves through the fistulous opening if there be one, and if not then the clove oil should be sealed in the canal, and in each case the clove dressing is allowed to remain a week, when in the great majority of cases the root filling may be proceeded with.

Root Filling.

As a root filling in these cases I have had excellent results from the use of gutta-percha, in which I have incorporated a little iodoform and hydronaphthol dissolved in eucalyptol. I use it quite thick and fill the chamber proper with gutta-percha. The tooth cavity may be filled with any material suitable to the case.

Sensitive Dentine.

As a means of doing away with the sensitiveness and at the same time stopping the progress of caries in shallow cavities, silver nitrate is very efficacious.

Cleaning Teeth.

It is important to keep children's teeth clean, and as often as tartar or green stains appear they should be removed. The removal of calculus and polishing the teeth is a very simple matter for children.

A little hydrogen dioxid added to pumice stone will aid in removing green stains. As a general rule it is never advisable to keep children in the dental chair longer than a half hour, nor is it advisable to adopt heroic painful measures when it can possibly be avoided even by a long way around.

By diverting the attention of a child we can often do quite painful things and have them bear it nicely; by this I do not mean that it is ever permissible to deceive a child. Once deceive a child he will always remember it and will *never trust you or any other dentist again.* By put-

ting into child phrases the condition present, and what you are trying to do for his relief and future comfort, and telling him some good stories of bravery and heroic deeds, and incidentally getting him interested in your imaginary rabbits, chickens, etc., you will accomplish what you desire and he will bless you always.

Management of Permanent Teeth During Childhood.

Very little needs to be said on this subject, because the various pathological conditions have already been presented either in this chapter or in the preceding ones. Happily the diseases of the gums and peridental membrane are rare at this period. I only wish to state that the most critical time for the permanent teeth is between the ages of six and fourteen, during the early period of which many first molars are lost. Parents as a rule regard this as a temporary tooth, and before the dentist has an opportunity to correct their mistake many teeth are hopelessly ruined.

In the chapter on capping pulps reference was made to the desirability of keeping pulps alive until the roots are fully developed. When the pulp is gone all further development of dentine ceases, and teeth whose roots are only partially formed cannot be expected to do a lifetime service, therefore every effort should be made to preserve the pulp, and when pulps are lost before the roots are nearly completed it is better to extract early, especially in six year molars, with the hope that the space will be occupied by the second molar by and by.

Management of Sensitive Cases.

While the usual obtundant remedies act well with children, it must be borne in mind that teeth are more sensitive during this period than later in life. Sensitive to malleting in fillings and also to excavating dentine, and that growing, maturing children cannot bear pain as well as older people, and yet if you have their confidence it is surprising what they will endure.

This is a period when it is difficult to get children to take that interest in the care of their teeth that they should, and consequently good mouth hygiene is often lacking, and as a result decay is more prevalent.

It is often wisest for all these reasons not to attempt permanent gold fillings, but rather to carry the teeth along in comfort with the use of the plastics until the time comes when all these conditions change and permanent results can be hoped for.

CHAPTER XXII.

Facial Neuralgia.

Etiology. Neuralgic Pains of Dental Origin. Causes. Symptoms. Diagnosis. Treatment. Resection.

The term neuralgia is derived from the Greek, and signifies nerve pain, and may be defined as "a painful affection of the nerves, due either to functional disturbance of their central or peripheral extremities or to neuritis in their course."—Osler.

Facial neuralgia is a term used to designate neuralgic pains in the region supplied by the fifth cranial nerve, sometimes called trifacial and trigeminal neuralgia.

Etiology.

Individuals who suffer from any chronic nervous disorder are most liable to this affection. Women more liable than men, and syphilitic, gouty, diabetic or anemic persons are most liable. In malarial districts the disease is very prevalent. Any irritation, especially if long continued, to any sensory nerve filament may be reflected in other sensory nerves, and if long enough continued may result in permanent neuritis.

The trifacial nerve arises by two roots, a small motor root and a large sensory root, upon which is situated the gasserion ganglion. Passing out from the ganglion are three main branches. First, the ophthalmic, which with its branches supplies the eye muscles, lachrymal gland, frontal muscles, eyelids, the nose; second, the superior maxillary, which with its branches supplies the upper jaw, the teeth, the orbit, the cheek; third, inferior maxillary, which through its terminal branches supplies the lower jaw, the teeth and some of its branching filaments pass to the ear and temporal region.

It readily can be seen, therefore, that any irritation to one of these branching filaments may be reflected in any branch of all three main divisions, and there is possibility of certain pains being transferred to another center in the brain through the anastomosing branches of other trunk nerves.

Neuralgic Pains of Dental Origin.

The dentist is mostly interested in pains arising from the dental organs or reflected to them. Sometimes these reflexes may be either or both motor and sensory, manifesting itself in pain and motor twitching and spasms.

Causes.

Facial neuralgia may develop from a variety of causes, both local and constitutional, as previously stated. When local the cause frequently lies within the tooth pulp or peridental membrane. Hyperemia of the pulp is sometimes the exciting cause. Pulp nodules impinging on the nerve filaments, all of which is contained with the walls of the pulp canals, may be an exciting cause; a case in point has been cited in Chapter IV. Uncovered sensitive dentine may transmit neuralgic pains.

Hypercementosis is sometimes considered a cause; septic diseases of the pulp have also been considered a cause.

Impacted teeth, particularly lower third molars, spicula of bone left after extraction, may impinge on the nerve. Ill-fitting lower dentures may press on the mental foramen, tumors in the bone, aneurysms, tumors of the nerves are frequent local causes.

Other causes which lie outside of the strictly dental organs are catarrhal conditions of the frontal or of the maxillary sinuses, inflammatory conditions about the eye and ear, and most important of all, inflammation of the nerves of the region affected, impingement of nerves in cicatrics about the jaws, following surgical operations.

All of these act more or less severe, according to this idiosyncrasy of the patient and the peculiar constitutional conditions present. After all has been said regarding these causes it must be stated that the more severe forms of facial neuralgia do not come from these sources, but in all probability arise from the nerve trunk itself or in the brain cells.

That most horrible of all forms, known as tic-douloureux, cannot be said to be caused by any condition associated with the teeth, unless it be as a mere starting point, from which it rapidly passes into serious trunk nerve disturbances, if, indeed, the brain cells are not the source. (Many think anemia of the nerve trunk is the cause.)

About this particular neuralgia we know very little except its manifestations. It produces the most excruciating pain; its attacks are paroxysmal, occurring with ever-increasing frequency until life becomes absolutely unendurable. The sight of the suffering of even one of these unfortunate victims will make a lasting impression on the observer.

When due to other dental causes than these there is never any constancy regarding the location of the pain; sometimes it will appear in one spot and sometimes in another; tenderness of the eyeball, the temporal and anterior auricular region, will usually indicate the trouble to be in the same part of the inferior maxillary branch.

Sometimes pains deflected to the ear, to the mastoid cells, in the infra-orbital or mental foramen region (Dr. Brophy has recited some inter-

esting cases of this character) have their origin in the lower teeth or jaw.

It is very rare to find neuralgia of dental origin affecting more than one of the trifacial branches at a time; the one most commonly affected when of dental origin is the inferior maxillary; when the upper teeth are responsible the pain may be deflected to the lip, nose or the cheek.

Symptoms.

There are very few dependable symptoms, although certain symptoms may serve as guide posts to direct the practitioner to the source. Before the onset of the pain there may be a peculiar tingling sensation in the part. The pain is not constant like from a forming abscess, but paroxysmal darting pains, twitching of the muscles of the part is usual.

The spasms come and go sometimes at regular intervals, and if it be a true neuralgia of trophic affection the pain will become severe, and even the skin may become so sensitive that the slightest touch will cause the sufferer to cry out with the pain.

If the pain be reflex and due to dental origin usually there is some discomfort about the mouth, or a careful examination as to caries, inflamed pulps, pericementitic erosions and the like will reveal the cause. As stated before, when the teeth themselves are responsible, it usually can be easily found either hot or cold sudden changes, recumbent position, tenderness to percussion or some recognizable disturbance can be seen.

When due to other than these peripheral causes the nerves will be tender to pressure at the points where they emerge from the bone.

Diagnosis.

The first essential in making a diagnosis of this trouble is to get a complete history of the case, even to the minutest detail; the patient may be able to give you a clue to the real trouble. The actual diagnosis must be made by exclusion; examine each tooth on the affected side for every known lesion, and, if none is found, the X-ray may be helpful in locating hypercementosis, pulp, nodules or impacted teeth, as well as tumors.

Next the region of the affected nerves should be examined for tender spots, which in tic-douloureux are located at the supra-orbital foramen, the upper eyelid, the cartilage of the nose, the parital eminence, when the ophthalmic branch is affected. The infra-orbital foramen, malor bone, upper lip, palate or other places in the upper jaw will be tender, when the superior maxillary is affected, or the tender spots may be in front of the ear, over the inferior dental foramen or over the mental foramen when the inferior maxillary branch is affected.

Treatment.

If any dental lesion exist it should be put in order, and thrice happy you should be if in doing so the source of the trouble has been found. It must be remembered that these pains sometimes continue for a few days after the exciting cause has been removed, but will gradually grow less and less.

Many therapeutic agents have been suggested as available in the treatment of facial neuralgia, among which are the following:

Phenacetine acetanilid in doses from five to ten grains in neuralgic pains about the face due to exposure to cold and dental irritation. Aconite tincture in five drop doses every twenty minutes will usually help the acute forms; it is administered until numbness of the lips appear; it is also used as a lotion painted liberally over the affected parts.

Arsenic in the form of Fowler's solution is especially recommended for neuralgias of malarial origin. It is best to begin with about ten minims and gradually decrease until one minim is given, then gradually increase again.

Butyl-chloral hydrate is strongly recommended in doses of about five grains. Many seem to rely on this drug. A mixture of butyl-chloral hydrate and tincture of camphor may be locally applied.

Belladonna is very useful if violent spasms are present.

Gelsemium in the form of the tincture and also the sulphate gelsemine has been found by the author to be very efficacious when the neuralgia is of dental origin.

Colchicum is a valuable remedy when the neuralgia is of gouty origin, best given in form of the wine of colchicum root, dose 5-20 minims.

Cannabis Indica has been praised as a remedy by many writers, but the author has had very negative results.

The opiates can never be considered as curative, but they are often our only means of controlling the severe pain. Many recommend injecting morphine directly into the affected region.

Cod liver oil and phosphorus has proven very efficacious in four cases treated by the author.

Iron and quinine are given often with helpful results when neuralgia is due to anæmia and malaria.

Local application of freezing mixtures such as methyl-chloride sprayed over the affected region has been helpful in some cases.

Electricity is considered by many very helpful. It must be carefully applied, using the positive electrode over the seat of the trouble. The current should be increased gradually and continued for twenty minutes at a time and repeated daily.

Castor oil treatment has been highly praised by many neurologists. The plan seems to be to give as much castor oil as possible without purgative effect. Dr. Patrick suggests that it is best given at bedtime. The first night a large dose is given which will purge considerably; the next night the same dose will purge less. This plan is followed for three or four days, when an additional dose may be given in the morning without purgative effect, and thus gradually the patient can take from two to four ounces in twenty-four hours without active purgation and at this point the real benefit begins.

The same author says that he has found this treatment beneficial in 40 per cent of the cases and curative in a somewhat smaller percentage.

In the treatment of these cases it is wise to try all of these means of relief before attempting the surgical methods, for the reason that even surgery fails to permanently cure many of these severe cases.

I have known of several cases where extraction of one tooth after another until all on the affected side were removed with only temporary benefit, and others where removal of the nerves in both jaws brought relief only for a few weeks. A great variety of surgical operations have been tried in times past for the relief of this trouble, all but four of which have been discarded.

The removal of the Gasserion ganglion promises the most permanent results, but even this has failed, and lately a suggestion has come from Abbe to sever the maxillary branches from the ganglion and place a piece of gutta-percha between the severed ends, thus preventing any future reunion. This operation is fraught with grave dangers to life and does not promise a permanent cure in all cases.

A German scientist has recently suggested injecting into the ganglion a one-half per cent solution of osmic acid. It seems to have proved successful in a few cases.

Resection.

Resection, an operation by which a portion of the affected nerve is removed. If the affected portion is removed before the entire trunk is involved success will follow, and in any event the patient will be free from pain for a period from two to six years.

This operation is simple compared with that of removing the ganglion, and if skillfully done is not dangerous. Most surgeons recommend that this operation be tried first, and if necessary the ganglion removed later.

Subcutaneous division, an operation by which the nerve trunk is divided, is successful for a short time; but my experience is that the pain returns with greater force and persistency.

Evulsion, an operation by which a portion of the nerve is torn out, is highly recommended by some surgeons, and by this method it is possible to tear out the nerve for a considerable distance, and thus bring freedom from pain for a considerable time.

A description of the technique of these operations does not come within the province of a work on therapeutics. The reader is referred to modern text books on surgical procedure.

CHAPTER XXIII.

Shock.

Etiology. Symptoms. Causes. Treatment.

Shock is a depression of the vital powers caused by injuries or from some great mental disturbance. It is manifest by a sudden check in the circulation brought about through the cerebro-spinal centers.

Etiology.

The condition follows accidents often in railway trains, or it may follow a profound mental impression or severe prolonged mental strain. It is not necessary that there be some physical lesion in order to produce shock from accidents, indeed, some of the severe forms appear when no physical signs can be found.

Shock from dental and surgical operations sometimes result when there is little or no loss of blood and most frequently from prolonged sittings in the dental chair; not always on account of the pain, but most frequently as a result of long mental strain from dread or fear.

Shock is not to be confounded with simple fainting, in which there is also a cessation of vital functioning. Shock may or may not appear for some time after accidents or operations. The usual history when caused by dental operations is, first, a feeling of over-excitement, which gradually passes into prostration, which may last for several days; and a few cases are reported where patients failed to rally and death resulted.

Symptoms.

The symptoms of shock usually begin by a tired feeling and appearance of prostration, and if profound the patient passes into a state of coma, where consciousness can scarcely be aroused, pallor of the face and the whole body surface, which is especially seen in the lips. The body is cold and covered with sweat, the eyelids droop, the features look pinched or the eyes in severe cases remain wide open and staring and have a weird and uncanny sunken look. The pulse is almost imperceptible, very weak and thready. The thermometer will show a temperature of 96° or 97°, respiration is short and feeble, or may be panting.

In these cases there is usually no great loss of sensibilities. Sometimes there will appear marked hysteria.

Causes.

Whatever the source of shock may be, it produces heart disturbance through the vaso motor system; there is a partial paralysis of this system, with some real cell injury which at present is not thoroughly understood.

Many regard shock as a temporary paresis of the muscles of the heart, but there evidently must be something more, and is probably explained by the theory of molecular nerve cell disturbance. Prognosis of shock is always uncertain. Of course the severity of the form will have much to do with the outcome.

Treatment.

The treatment must always be based on the severity of the various symptoms. The recumbent position is essential. As soon as possible warm stimulating drinks should be given; whiskey or brandy are common remedies which are valuable. Volatile heart and respiratory stimulants, such as amyl nitrate and ammonia, should be held before the face to tide over the temporary vital depression.

The slapping of the face with a cold wet towel and the chafing of the extremities are helpful especially in the cases of syncope or fainting. Then artificial respiration should be undertaken and kept up as long as it is helpful.

Hypodermic injections of atropine to maintain the respiration and nitro-glycerine, one two-hundredth of a grain. Digitalis and strychnine, one-twentieth of a grain, are the remedies to support the circulation.

When hysterical excitement prevails, morphine one-eighth to one-fourth of a grain should be given. When the patient can readily swallow, the aromatic spirits of ammonia is a valuable remedy, as well as valerian.

In concluding this article I wish to call attention to the danger of shock from severe prolonged dental operations. The symptoms may not appear at the time, but may develop several hours or days after the sitting. Long sittings should be avoided, especially if patients are nervous or excitable.

It is better to make two or more short sittings, or if necessary make only temporary operations rather than run the risk of inducing disturbances which are fraught with such grave dangers.

The medicine case should always be supplied with the usual remedies indicated in these cases, for oftentimes they will be called for on a moment's notice.

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